STUDY OF LUNG DISEASES IN BUNDELKHAND REGION OF U.P. (A POSTMORTEM STUDY)

THESIS

FOR

DOCTOR OF MEDICINE

(PATHOLOGY)

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JHANSI (U.P.)

2001

CHANDRA BHAL SINGH

CERTIFICATE

This is to certify that the work entitled **"STUDY OF LUNG DISEASES IN BUNDELKHAND REGION OF U.P. (A POSTMORTEM STUDY)"** being submitted for M.D. (Pathology) has been carried out by **DR. CHANDRA BHAL SINGH** himself in this department.

He has been put the necessary stay in the department as required by the regulation of Bundelkhand University Jhansi.

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This work fulfils the basic ordinances governing the submission of thesis laid down by Bundelkhand University, Jhansi.

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Date : 27.11.2000

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(CHANDRA BHAL SINGH)

CONTENTS

INTRODUCTION

INTRODUCTION

The lung diseases form a large percentage of attendance in any general hospital in our country as well as in other countries of the world. People suffer from various infections of the upper respiratory tract and lungs. The number of people suffering from pulmonary tuberculosis is high. The non-tubercular pulmonary diseases are recognized in greater number with the use of newer methods of diagnosis viz. Bronchial biopsy, FNAC, Bronchoalveolar lavage (BAL) specimen, cytological examination, special stainings, electron microscopy, Immunohistochemistry, Enzyme Linked Inmmnosorbent Assay (ELISA) and polymerase chain reaction (PCR) etc. (Anthony P. et al 1987)

There is increased incidence of pneumonias (10-20% in developing countries- Park, 1997) chronic obstructive pulmonary diseases (COPD) and chronic obstructive airway diseases (COAD) such as emphysema, chronic bronchitis, bronchial asthma, bronchiectasis and other lung diseases like pulmonary tuberculosis and various dust diseases- Pneumoconiosis, due to great urbanization, Industrialization, environmental pollution and increased cigarette smoking habits including passive smoking.

Pulmonary tuberculosis is still a single most important chronic bacterial infection world wide specially in India where

prevalence of infection is about 30% (35% in males and 25% in females). Currently the incidence rate of pulmonary tuberculosis for India is 1-2% (W.H.O. bulletin, 1997). The number of cases of any one time has been estimated to be at least 1.5% of the population suffering from radiologically active tuberculosis with about one fourth of the cases being sputum positive or infection. It is estimated that there are 500,000 deaths annually due to pulmonary tuberculosis , while a similar number of persons get cured. This is more than balanced by an addition of about 2-2.5 million sputum positive cases annually (Park, 1997).

The variety of benign and malignant tumors may arise in lung but the vast majority are Bronchogenic carcinoma accounts for 90-95%.

The pulmonary neoplasm now account for third most common cancer in India where its incidence is 6.3% of total malignancies. It is the most common cancer among males in India with incidence of 10.6% of all malignancies. The age specific rate for lung cancer is estimated to be 10.4 per 100,000 for males and 1.6 per 100,000 for females (Rao et al, 1998).

Recently there has been increase in mortality and morbidity due to respiratory diseases specially lung cancer in both developed as well as in developing countries. The age adjusted death rate attributable to lung cancer in men

increased by 121% and by 425% in women between 1956 to 1990 (Anderson, 1996). The incidence of deaths from lung carcinoma is higher in urban areas and an urban factor is incriminated in the form of atmospheric pollution of town air with exhaust fumes ; dust, smoke and smog.

The extent of discrepencies between clinical diagnosis and autopsy diagnosis varies widely from study to study. In compilations by Nemetz et al (1987) and wheeler (1987) the possibility of a missed clinical diagnosis ranged from as little as 4% to as much as 66%. In this study Cechner et al (1980) studied 415 deaths in series, out of which 28% clinically occult bronchogenic carcinoma discovered at autopsy.

Neonatal autopsy study has been revealed 10-15% cases of pulmonary hypoplasia (Anderson, 1996).

Emphysema has been a common finding at autopsy with a prevalence of 20-100% depending on the population studied and the technique and criteria used (Anderson, 1996).

The sequelae of embolism in the pulmonary arterial tree are often seen at autopsy in 50-60% of hospital deaths (Anderson, 1996) specially in elderly and people who have been confined to bed with congestive heart failure and in adults of any age who have undergone a major surgical operation. Pulmonary embolism also occurs after parturition. Pulmonary emboli are primarily responsible for nearly 5-10

cases of all hospital deaths in western countries (Mittal et al, 1998). Various pneumonias are frequently missed at autopsy or mistaken for pulmonary embolism. The reverse is also true (Anderson, 1996).

One retrospective autopsy study between Jan 1982 and Dec. 1992, of 10037 autopsies performed, 0.87% patients had died due to bleeding diathesis, out of which lungs were involved in 20 cases (Bhatia et al, 1998).

An autopsy study carried out in 92 AIDS patients in India between 1989 and 1996, has shown tuberculosis in 64 patients , CMV infection in 3 patients and Cryptococcus neoformans infection in 4 cases, where lungs were involved (Lanjewar et al 1998).

The present day knowledge of medicine largely dependent upon Autopsy / Necropsy studies (Rokitansky, 1804-1878, who performed 3000autopsies).

The certain lung diseases which were discovered at autopsy includes : Legionnaire's disease and related pneumonias, Hantavirus lung diseases, occupational lung diseases, pulmonary embolism, diffuse interstitial fibrosis of lung etc. (Hill & Anderson, 1996).

Literature testifies to the rarity of such kind of work ever conducted in this part of country specially in Bundelkhand region of U.P. and hence opportunity of taking up the present study. The autopsy study was aimed to assess the incidence and pattern of different lung diseases in Bundelkhand region of U.P. in persons dying of accidental deaths and otherwise.

MATERIAL AND METHODS

MATERIAL AND METHODS

The present study was conducted in the department of pathology, Maharani Laxmi Bai Medical College and Hospital Jhansi to assess the incidence and pattern of different lung diseases by autopsy examination. Material for study included lung specimens for the study which were collected from the dead bodies brought from different parts of Bundelkhand region for the post mortem examination in the mortuary of M.L.B. Medical College and Hospital , Jhansi.

Post Mortem Examination

Dead bodies were opened by a mid line incision from just above thyroid cortilage to the pubic symphysis, avoiding the umblicus and any injuries in the line of incision (I-shaped incision method). Modified 'Y' shaped incision was made specially in females (Parikh, 1992)

Lung specimens were collected from the dead bodies during post mortem examination and kept in large plastic containers using 10% formalin for fixation and preservation. Tissues collected were subjected for following :-

a. Gross examination :

Gross examination and dissection was carried out in the pathology department. Tissue sections of reasonable

thickness were passed from representative areas of the lung specimens, both Right and Left.

b. Histopathological examination :

Tissue sections were subjected to process of dehydration, clearing and paraffin embedding technique to prepare paraffin blocks.

Section Cutting -

These paraffin blocks were cut into thin sections ranging from 4-5 μ in thickness on microtome.

Staining -

These sections were subjected for -

1. Routine Haematoxylin and Eosin (H&E) staining method (Culling,1975) for microscopic examination.
2. Other sections were subjected to special staining wherever needed like :-
    1. Periodic Acid schiff stain (PAS) for morphological staining of basement membranes and mucoid substances (Culling, 1975).
    2. Southgate's mucicarmine for epithelial mucin (Culling, 1975).
    3. Alcian blue for connective tissue mucin (Culling, 1975).

4. GORDON & SWEETS Silver Impregnation method for reticulin fibres (Culling, 1975).

5. Mallory's Phosphotungstic Acid Haematoxylin stain (P.T.A.H.) for fibrin (Culling, 1975).

6. Verhoeff's staining for elastic tissues (Culling, 1975).

Haematoxylin & Eosin Staining Procedure (Culling 1975).

1. Sections were deparaffined and brought to water through graded solutions of alcohol.
2. Sections were stained in a solution of Harris haematoxylin for 5-15 minutes.
3. It was followed by washing in running water till sections turned blue.
4. Sections were decolourised with one percent acid (HCl) alcohol solution.
5. Washed in running water for 5-15 minutes till sections again turned blue.
6. Counter stained with one percent aqueous Eosin for 1 minute.
7. Washed rapidly in water and blotted.
8. Dehydrated in several changes of 70%, 80%, 90% and absolute alcohol.
9. Cleaned in xyline and mounted in D.P.X.

All the informations regarding patients/victims illness , clinical data, pathological findings and observations were recorded on a preset proforma for further analysis.

WORKING PROFORMA

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POST MORTEM DATA

Serial No..... Post Mortem Record No.....

Date & Time of postmortem.....

Name of deceased :..... Age\sex :.....

Time of since death :.....Ward\Bed No.....

Place of death .....

Mode\Nature of death .....

Clinical Diagnosis, if any .....

Any other relevant information .....

POST MORTEM FINDINGS

Body :

Primary incision:

pleural cavities:

Mediastinum :

Pericardial cavity :

LUNGS Weight Right Left.

AUTOPSY \PATHOLOGICAL DATA :

Gross examination of organs (Lungs):

Size : Colour :

Bronchial Markings :- Any Other Fingdings :-

Section :-

Staining : - Routine H & E staining :

Special stainings :- Verhoeff PAS PTAH V.G.

MICROSCOPIC FINDINGS

Clinicopathologicl Correlation :-

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The respiratory system develops from a median diverticulum of the foregut at about 4-6 weeks of gestation. At this time from lung buds, bronchi and lung parenchyma develops. Further subdivision of the lobar bronchi results in the development of lung substance. The lungs are covered by pleura and fissures separates the lobes. Gas exchange sites and surfactant system develops at the end of 27th weeks of gestation , necessary for sustained extra-uterine respiration (Anderson, 1996).

There are two lungs right and left. Adult right lung weighs 375 to 550 gm (average 450 gm) has three lobes with 10 bronchopulmonary segments whereas adult left lung weighs 325 to 450 gm (average 400 gm) has two lobes with 8 bronchopulmonary segments. The lung consist of airways, blood vessels, connective tissue frame work and the pleura. The airways begins as the left and right major bronchus which further divide into bronchi and terminal bronchioles before the first respiratory bronchioles are reached. The part of the lung tissue distal to terminal bronchiole is called an acinus. A cluster of about 5 acini enclosed by visible fibrous septa form pulmonary lobule. Trachea and bronchi are lined by pseudostratified coloumner epithelium while simple coloumner epithelium lining the bronchioli is composed

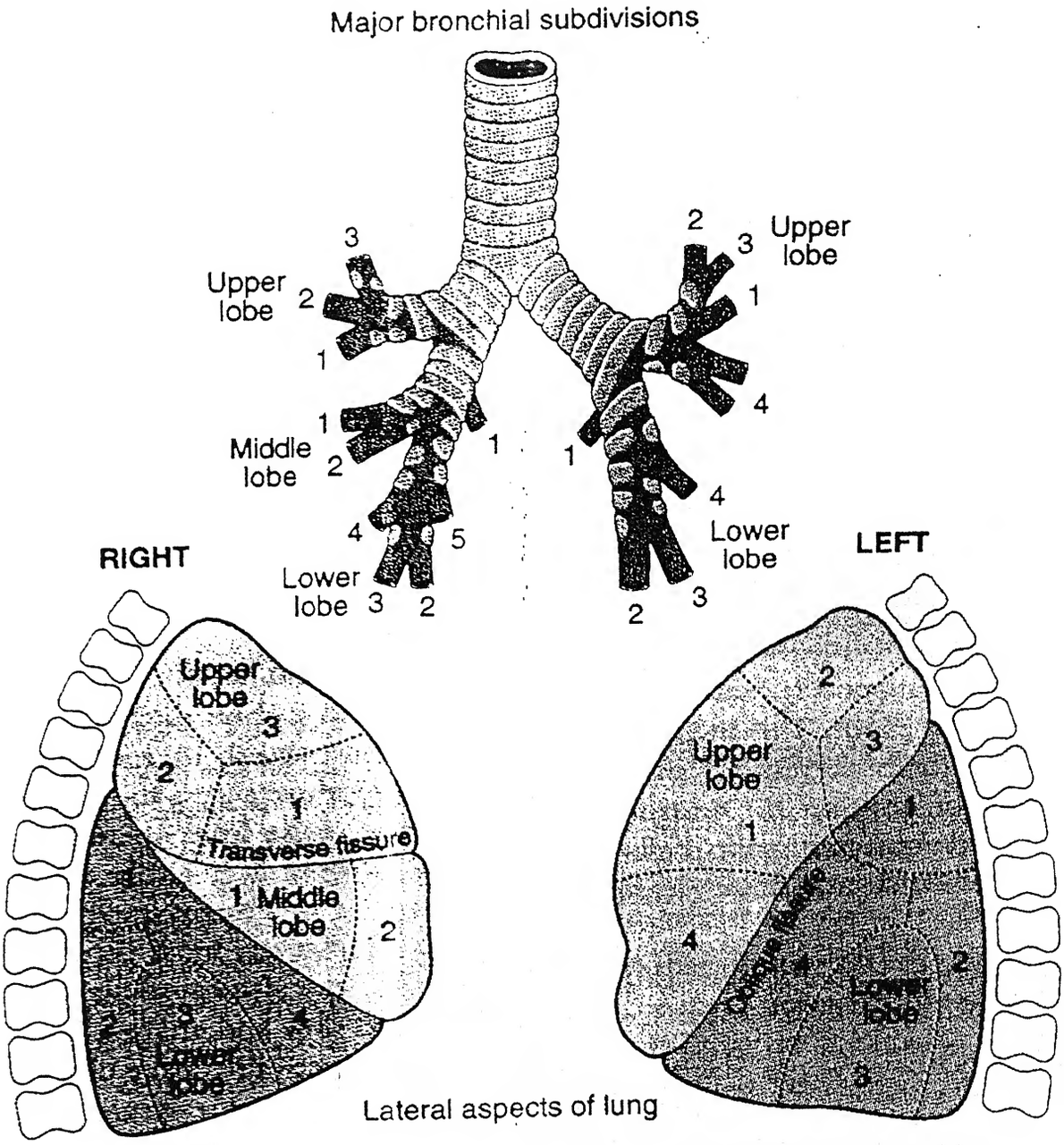


Fig. 1 - The Major bronchial divisions and fissures, lobes and segments of the lungs. (Davidson)

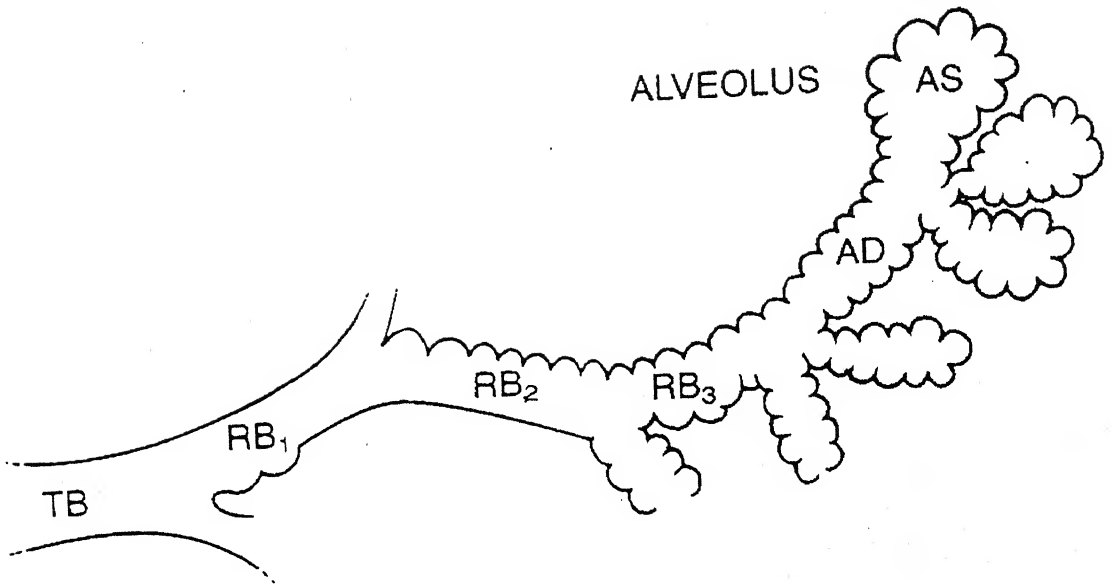


Fig. 2 - The structure of an acinus. (Robbins)

TB - Terminal bronchiole
RB_1 - Respiratory bronchiole of first order
RB_2 - Respiratory bronchiole of second order
RB_3 - Respiratory bronchiole of third order
AD - Alveolar duct
AS - Alveolar sac.

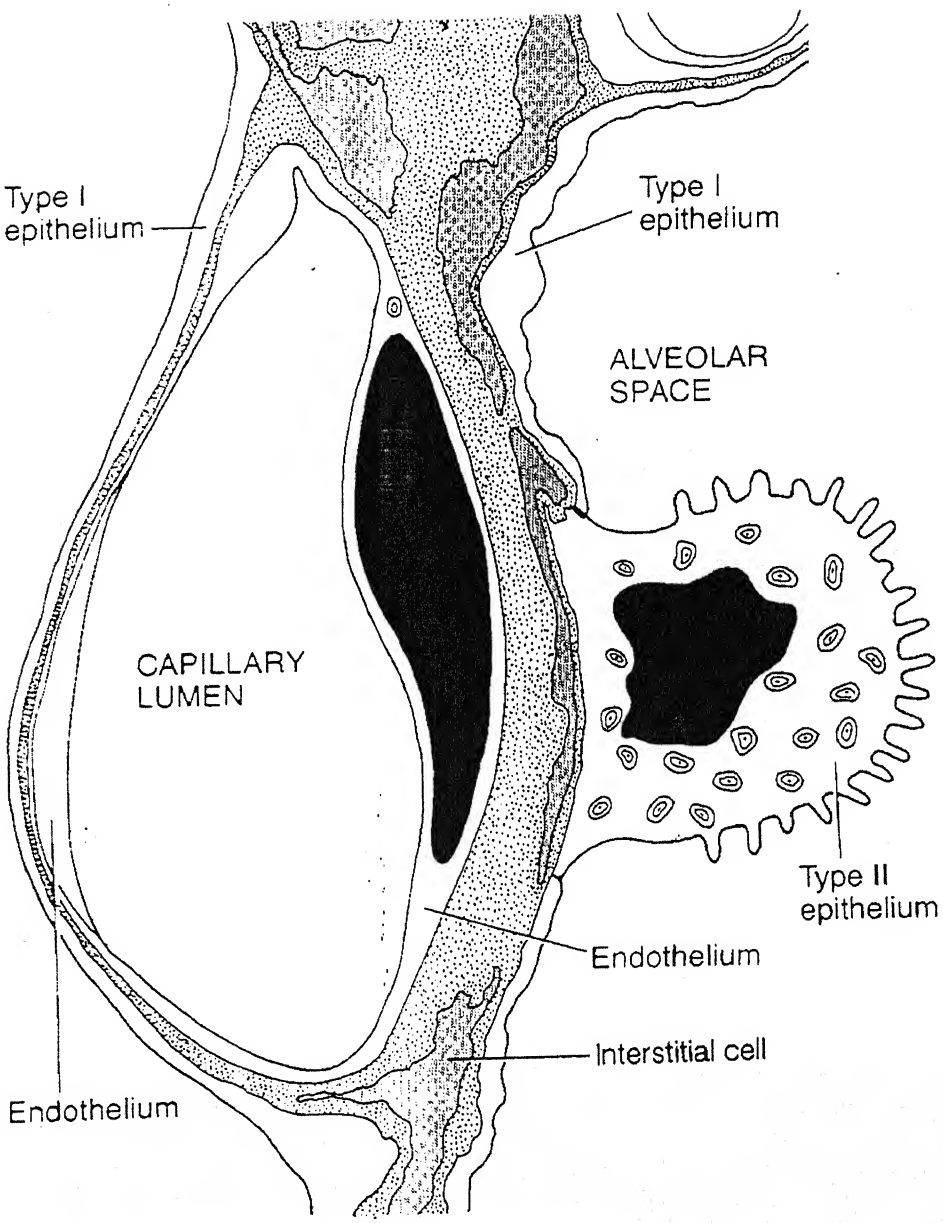


Fig.3-Microscopic structure of alveolar wall.(Robbins)

mainly of ciliated epithelial cells interspersed with non-ciliated secretary cells called CLARA CELLS. Respiratory bronchioles bear several alveoli on their walls and it may also divide into several alveolar ducts which open in to4-5 atria bearing many alveoli. The alveolar lining predominantly consists of type 1 pneumocytes. Type-II pneumocytes are also interspersed in between. The acinus is the basic gas exchange unit of lung (Anderson, 1996) [Fig-1 (Davidson), Fig 2,3 - (Robbins).

The primary function of lung is oxygenation of blood and removal of carbon - dioxide. Other functions of lungs are to maintain acid base balance and water evaporation which is an important factor in the fluid balance and heat regulation of the body. The cardio-vascular and renal system are inter-related with pulmonary function.

Different type of lung diseases may be classified according to **Hanshaw & Murray (1980)** -

1. Developmental Abnormalities
   - (i) Agenesis, aplasia and hypoplasia of lung.
   - (ii) Hyaline membrane disease.
2. Infectious Diseases of the lung :
   - (i) Respiratory infection due to viruses, mycoplasma, chlamydia etc.
   - (ii) Bacterial pneumonias :
     - a. Bronchopneumonia.

b. Lobar pneumonia.

(iii) Lung Abscess

(iv) Tuberculosis

a. Primary Tuberculosis

b. Secondary (Reactive) Tuberculosis.

(v) Actinomycosis and Nocardiosis

(vi) Fungal diseases

a. Histoplasmosis

b. Coccidiodomycosis

c. Blastomycosis

d. Other pulmonary mycosis.

(vii) Parasitic diseases in the compromised host.

3. Neoplastic diseases

a. Benign

b. Malignant

c. Metastatic.

4. Disorders of Airways :

(i) Asthma

(ii) Chronic bronchitis and Emphysema

(iii) Bronchietasis and cystic fibrosis

(iv) Bronchial obstruction, Atelectasis & Broncholethiasis.

5. Disorders of pulmonary circulation

   (i) Pulmonary edema

   (ii) Pulmonary thrombo embolism.

   (iii) Pulmonary hypertension and corpulmonale.

6. Pulmonary diseases of Environmental, physical or chemical origin.

   (i) Pneumoconiosis and related diseases

   (ii) Occupational Asthma, Byssinosis and other occupational lung diseases.

   (iii) Chemical,radiation,thermal and aspiration injuries.

   (iv) Thoracic trauma.

7. Diffuse diseases of the lung of unknown origin -

   (i) Sarcoidosis

   (ii) Idiopathic pulmonary fibrosis, Lymphoid interstitial pneumonias, hypersensitivity pneumonias and collagen vascular diseases.

   (iii) Histiocytosis X, Hemorrhage Syndrome and other rare diffuse infiltrative lung diseases.

   (iv) Drug induced respiratory disorders.

8. Disorders of pleura, diaphagm and mediastinum.

Lung tumors are histologically classified according to W.H.O. classification (1981).

W.H.O. Classification of Lung Tumors (1981)

I. EPITHELIAL TUMORS OF THE LUNG :

A. BENIGN

1. Papillomas

a. Squamous cell papilloma

b. Transitional papilloma

2. Adenomas

a. Pleomorphic adenoma ('mixed' tumor)

b. Monomorphic adenoma

c. Others

B. DYSPLASIA / CARCINOMA IN SITU :

C. MALIGNANT

1. Squamous cell carcinoma (Epidermoid carcinoma) variant : spindle cell (squamoid) carcinoma.

2. Small cell carcinoma

a. Oat cell carcinoma

b. Intermediate cell type

c. Combined oat cell carcinoma.

3. Adenocarcinoma

a. Papillary adenocarcinoma.

b. Acinar adenocarcinoma

c. Bronchiolo-alveolar carcinoma.

d. Solid carcinoma with mucus formation.

4. Large cell carcinoma

Variants a. Giant cell carcinoma

b. Clear cell carcinoma

5. Adenosquamous carcinoma.

6. Carcinoid Tumor

7. Bronchial gland carcinoma.

a. Adenoid cystic carcinoma.

b. Mucoepidermoid carcinoma

c. Others

8. Others

II. SOFT TISSUE TUMORS PRIMARY IN THE LUNG

III. PLEURAL TUMORS

a. Benign mesothelioma

b. Malignant mesothelioma.

IV. MISCELLANEOUS TUMORS

a. Benign tumors

b. Malignant tumors

(i) Carcinosarcoma

(ii) Pulmonary blastoma

(iii) Malignant melanoma

(iv) Malignant lymphoma

(v) Others

V. SECONDARY TUMORS

VI. UNCLASSIFIED TUMORS

VII. TUMOR- LIKE LESIONS.

i) Hamartoma

ii) Lymphoproliferative lesions

iii) Tumorlet

iv) Eosinophilic granuloma

v) Sclerosing Haemangioma

vi) Inflammatory Pseudotumor

vii) Others.

I. Developmental Abnormalities

1. Pulmonary Hypoplasia

Incomplete or defective development resulting in an overall decrease in relative volume or lung weight because of reduction in the number or size of acini.

2. *Hyaline Membrane Disease : (HMD)*

HMD is a clinical and pathological perinatal disorder related to the deficiency of pulmonary surfactant.

Grossly ; the lungs are normal in size, reddish purple in colour , solid and airless.

Microscopically ; the presence of collapsed alveoli ; (atelactasis) alternating with dilated alveoli , formation of characteristic eosinophilic hyaline membranes lining the respiratory bronchioles, alveolar ducts and the proximal alveoli, vascular congestion, focal hemorrhages and dilatation of septal lymphatics and absence of inflammatory reaction.

II. Infectious Diseases of the lung :

1. *Pneumonia*

It is acute inflammation of the lung parenchyma distal to the terminal bronchioles having incidence of about 10-20% in developing countries. Bacterial infection of the lung parenchyma is the most common cause of pneumonia or consolidation of one or both the lungs.

A. *Bacterial Pneumonias*

1. *Lobar Pneumonia* -

It is an acute bacterial infection of a part of lobe, the entire lobe or even two lobes of one or both the lungs caused by streptococcus pneumoniae (More than 90% of all lobar pneumonias), Staphylococcus aureus, β-haemolytic strepto-

cocci, and less commonly by gram-negative bacteria like Haemophilus influenzae, Klebsiella pneumoniae, Pseudomonas, Proteus, Escherichia coli.

The lower lobes are affected most commonly. Four stages of the inflammatory response classically occurs i.e. stage of congestion, stage of red hepatisation, stage of gray hepatisation and stage of resolution. Often at autopsy the anatomic changes do not confirm to the older classic stages due to effective antibiotic therapy.

Grossly : The affected lobe is enlarged, heavy, dark red, congested and subcrepitant which later on becomes firm and consolidated. Initially cut surface exudes blood stained frothy fluid , later on cut surfae becomes airless, dry, granular and gray.

Microscopically : Initially engorgement, intra-alveolar fluid with few neutrophils and numerous bacteria demonstrated by Gram's staining. Later on dense and numerous fibrin strands and macrophages are present in the alveolar spaces.

2. *Bronchopneumonia (Lobular Pneumonia)*

Patchy consolidation of the lung is the dominant characteristic of bronchopneumonia ; usually represents an extension of a preexisting bronchitis or bronchiolitis ; more commonly occurs in infancy and old age. It is a common finding on postmortem examinations. The common organisms responsible for bronchopneumonia are Staphylococci, Strep-

tococci, Pneumococci, Hemophilus influenzae, Pseudomonas aeruginosa and Coliform bacteria.

Grossly : patchy areas of red or gray consolidation affecting one or more lobes , cut surface is dry , granular, firm, red or gray in colour , often centred around a bronchiole.

Microscopically : acute bronchiolitis, suppurative exudate, consisting chiefly of neutrophils in the peribronchiolar alveoli and thickened alveolar septa.

B. Viral and Mycoplasmal Pneumonia (Primary Atypical Pneumonia)

Characterized by patchy inflammatory changes, largely confined to interstitial tissue of the lungs, without any alveolar exudate. Interstitial pneumonitis is caused by a variety of organisms, the most common being Mycoplasma pneumoniae, other agents are influenza virus A and B, respiratory syncytial viruses (RSV), adenovirus, rhinoviruses, rubeola and varicella viruses, chlamydia and coxiella burnetii.

Grossly : Patchy to massive and widespread consolidation of one or both the lungs. The lungs are heavy, congested and subcrepitant, sectioned surface exudes small amount of frothy or bloody fluid.

Microscopically : Thickening of alveolar walls, edema, mononuclear inflammatory infiltrate, foci of necrosis of the bronchiolar epithelium , inspissated secretions in the lumen, multinucleate giant cells and syncytia in the bronchiolar and

alveolar walls, occasionally viral inclusions (intranuclear and / or intracytoplasmic) especially in CMV pneumonia, and hyaline membrane.

C. Other Types of Pneumonia -

Some other types of pneumonias caused by infective agents (usually in immunocompromised hosts) and certain non infective varieties.

(i). Pneumocystis Carinii Pneumonia (PCP) -

Pneumocystis carinii, a protozoa, causes pneumonia by inhalation of the organisms as an opportunistic infection in neonates and immunocompromised hosts. Almost 100% cases of AIDS develop opportunistic infection, most commonly PCP. Other immunosuppressed groups are patients on chemotherapy for organ transplant and tumors, malnutrition, agammaglobulinaemia etc.

Grossly ; lungs are consolidated , dry and gray.

Microscopically ; interstitial pneumonitis , pink frothy fluid in the alveolar lumen, GMS stain demonstrates characteristic oval or crescentric cysts , surrounded by numerous tiny black dot like trophozoites of P. carinii in the frothy fluid.

(ii). Legionella Pneumonia -

Legionella pneumonia or legionnaire's disease is an epidemic illness caused by gram negative bacilli, Legionella pneumophila. Impaired host defenses in the form of

immunodeficiency , corticosteroid therapy, old age and cigarette smoking play important roles.

Grossly : Widespread bronchopneumonia invalving many lobes, consolidation and frequently pleural effusion.

Microscopically : Intra alveolar exudate, foci of hyperplasia of the lining epithelium of alveolar septa, thrombosis of vessels in the septa ; organisms may be demonstrated in the macrophages by special stains or by immunofluorescent techniques.

(iii). Aspiration (Inhalation) pneumonia

Results from inhaling different agents into lungs includes food, gastric contents, foreign and infected material from oral cavity. Predisposing factors include unconsciousness, drunkenness, neurological disorders affecting swallowing, drowning, necrotic oropharyngeal tumors in premature infants and congenital tracheo-oesophageal fistula.

Aspiration of small amounts of sterile foreign matter such as acidic gastric contents produce chemical pneumonitis characterized by hemorrhagic pulmonary edema with presence of particles in the bronchioles. Non-sterile aspirate causes wide spread bronchopneumonia with multiple areas of necrosis and suppuration. Aspirated material may be surrounded by granulomatous reaction with foreign body giant cells.

(iv) Hypostatic Pneumonia -

It is a common terminal event in the old, feeble, comatose patients characterized by collection of edema fluid and secretions in the dependent parts of the lungs , may produce bacterial pneumonia.

(v). Lipid Pneumonia -

It is of two types : Exogenous lipid pneumonia caused by aspiration of variety of oily materials like nasal drops, liquid paraffin and oily vitamin preparation. Endogenous lipid pneumonia causing pneumonic consolidation due to tissue break down following obstruction to airways e.g. obstruction by bronchogenic cancer, tuberculosis and bronchiectasis.

Grossly - Consolidation of affected part of lung , cut surface is characteristically 'golden yellow'.

Microscopically - foamy macrophages within the alveolar spaces , cholesterol clefts, formation of granuloma with FB giant cells.

2. Lung Abcess -

It is a localized area of necrosis of lung tissue with suppuration. The commonly isolated organisms include aerobic and anaerobic streptococci, staphylococcus aureus and a host of gram negative organisms which introduced into the lungs from one of the mechanism like aspiration of

infective material (most common cause), preceding bacterial infection, bronchial obstruction, septic embolism etc.

Grossly ; abcess cavity may be of variable sizes, contains exudate.

Microscopically ; destruction of lung Parenchyma with suppurative exudate in the lung cavity, surrounded by acute inflammatory cells in the wall initially, later on replaced by exudate of lymphocytes,plasma cells and macrophages.

3. *Pulmonary Tuberculosis* -

Tuberculosis is a world wide, chronic communicable disease caused by "Koch bacillus", mycobacterium tuberculosis (slender, rod-like bacillus, stained by Ziehl-Neelson stain), which usually affects the lungs and other tissues of human body. Currently the incidence rate of pulmonary tuberculosis for India is 1-2%. "Caseating granulomas" are the histologic hallmarks of tuberculosis but tubercle bacilli should always be demonstrated to confirm the histologic diagnosis of the tuberculosis. Human beings acquire infection by Inhalation, Ingestion, Inoculation or by transplacental routes. The disease spreads in the body by various routes like local spread, lymphatic spread, haematogenous spread and by the natural passages. The pathogenesis of tuberculosis depends upon the virulence of M.tuberculosis, role of induced hypersensitivity, role of immunity or resistance and the genesis of the granulomatous

pattern of reaction. Tissue changes seen in tuberculosis are not the results of any exotoxin or endotoxin but are instead the result of host response to the organism , in the form of development of cell- mediated hypersensitivity(Delayed or Type IV hypersensitivity) and immunity.

Types of Tuberculosis

(a) *Primary Tuberculosis* :

Defined as infection of an individual lacking previous contact with tubercle bacilli. In primary lung infection, a single lesion (GHON FOCUS) is usually found immediately subjascent to the pleura in the lower part of upper lobes or upper part of the lower lobes of one lung. The combination of the primary lung lesion and lymph node involvement is referred to as the "GHON COMPLEX".

Grossly ; lesion in the lung is the primary focus or Ghon's focus ; 1-2 cm solitary area of tuberculous pneumonia located under the pleura , in the lower part of upper lobe.

Microscopically ; the lung lesion consists of tuberculous granulomas with caseation necrosis.

(b) *Secondary Tuberculosis -*

Secondary or post primary tuberculosis is that phase of tuberculous infection that arises in a previously sensitized individual , whether the tubercle bacilli are derived from endogenous or exogenous sources. The lesion usually begins

as 1-2 cm. apical area of consolidation of the lung followed by a small area of central cascation, necrosis and peripheral fibrosis. Patients with HIV infection previously exposed to tuberculous infection have particularly high incidence of reactivation of primary tuberculosis. In addition opportunistic infection with M. avium - intracellulare can occur in cases of AIDS. The course of apical infection is extremely varied.

(i) *Fibrocaseous Tuberculosis :*

The original area of tuberculous pneumonia undergoes massive central caseation, necrosis which may form cavitary or open fibrocaseous tuberculosis or remain as a non-cavitary lesion (chronic fibrocaseous tuberculosis).

Grossly ; tuberculous cavity is spherical with thick fibrous wall, lined by yellowish, caseous, necrotic material and the lumen is traversed by thrombosed blood vessels ; foci of consolidation around the wall of cavity, overlying pleura may be thickened.

Microscopically ; the wall of cavity shows eosinophilic, granular, caseous material which may show foci of dystrophic calcification. Widespread coalesced tuberculous granuloma composed of epitheloid cells, Langhan's giant cells and peripheral mantle of lymphocytes having central caseation necrosis and fibrosis of the outer wall of cavity.

(ii) *Tuberculous caseous pneumonia* :

The caseous material from a case of secondary tuberculosis in an individual with high degree of hypersensitivity may spread to rest of the lung producing caseous pneumonia.

Microscopically ; exudative reaction with edema, fibrin, polymorphs and monocytes, numerous tubercle bacilli can be demonstrated in the exudate.

(iii) *Miliary tuberculosis* :

It is lymphohematogenous spread of tuberculous infection from primary focus or later stages of tuberculosis.

Grossly ; the miliary lesion are millet seed - sized (1mm diameter), yellowish, firm areas without grossly visible caseation necrosis.

Microscopically ; structure of tubercles with minute areas of caseation necrosis.

C. Anonymous (Atypical) Mycobacterial infections :

Occasionally ,human tuberculosis may be caused by atypical mycobacteria which are non-pathogenic to guinea pigs and resistant to usual anti-tubercular drugs. The prevalence of atypical mycobacterioses is on the rise because of their association with AIDS. There are four groups of atypical mycobacteria.

Group I- Photochromogens, which produce yellow pigment in the culture grown in light.

Group II - Scotochromogens, produce pigment whether the growth is in light or in dark.

Group III- Non chromogens, produce no pigments and the organism is closely related to avium bacillus.

Group IV- Rapid growers, grow fast in culture but are less pathogenic than others.

The infection by atypical mycobacteria is acquired directly from the environment, unlike person-to-person transmission of classical tuberculosis and the disease produced is known as atypical mycobacteriosis. The lesions produced may be granulomas , nodular collection of foamy cells or acute inflammation. Five pattern of disease are recognized.

(i) Pulmonary disease produced by M. Kansasi or M. avium intracellulare.

(ii) Lymphadenitis caused by M. avium - intracellulare or M. Scrofulaceum.

(iii) Ulcerated skin lesion produced by M. ulcerans or M. marinum.

(iv) Abscess caused by M. fortuitum or M. chelonei.

(v) Bacteraemias caused by M. avium - intracellulare as seen in immunosuppressed patients of AIDS.

4. *FUNGAL DISEASES :*

Fungal infections of the lung are more common than tuberculosis in the USA. These infections in healthy individuals are rarely serious but in immunosuppressed individuals may prove fatal.

(i) Aspergillosis -

It is the most common fungal infection of the lung caused by Aspergillus fumigatus. The fungus exist as thin septate hyphae with dichotomous branching and grows best in cool, wet climate. The infection may result in allergic bronchopulmonary aspergillosis, aspergilloma and nercotizing bronchitis. Immunocompromised person develop more serious manifestations of aspergillous infection specially in leukaemic patients on cytotoxic drug therapy. Extensive hematogenous spread of aspergillus infection may result in widespread changes in lung tissue due to arterial occlusion , thrombosis and infarction.

(ii) Histoplasmosis -

It is caused by oval organism, Histoplasma capsulatum by inhalation of infected dust or bird droppings. The condition may remain asymptomatic or may produce lesion similar to the Ghon complex.

(iii) Coccidiodomycosis -

It is caused by coccidiodes immitis which are spherical spores. The infection in human beings is acquired by close contact with infected dogs. The lesion consist of peripheral parenchymal granuloma in the lung.

(iv) Cryptococcosis

It is caused by Cryptococcus neoformans which is round yeast having a halo around it due to shrinkage in tissue sections. The infection occurs from infection by inhalation of pigeon droppings. The lesion in the body may range from a small parenchymal granuloma in the lung to cryptococcal meningitis.

(v) Blastomycosis -

It is an uncommon condition caused by Blastomyces dermatitidis. The lesions result from inhalation of spores in the ground. Pathological features may present as Ghon complex like lesion, as a pneumonic consolidation and as multiple skin nodules.

(vi) Mucormycosis -

Mucormycosis or phycomycosis is caused by mucor and Rhizopus. The infection in the lung occurs in a similar way as in aspergillosis. The pulmonary lesions are specially common in patients of diabetic ketoacidosis.

(vii) Candidiasis -

Candidiasis or moniliasis caused by candida albicans is a normal commensal in oral cavity, gut and vagina but attains pathologic form in immnocompromised host. Angioinvasive growth of the organism may occur in the airways.

III. Chronic obstructive pulmonary Disease :

Chronic obstructive pulmonary disease (COPD) and chronic obstructive airway disease (COAD) refer to a group of conditions - chronic bronchitis, emphysema, bronchial asthma and bronchiectasis ; that are accompanied by chronic or recurrent obstruction to air flow within the lung. Because of the increase in environmental pollutants, cigarette smoking and other noxious exposures, the incidence of COPD has increased dramatically in the past few decades.

1. Chronic Bronchitis :

It is a common condition defined clinically as persistent cough with expectoration on most days for at least three months of the year for two or more consecutive years. It is most frequent in middle-aged men. 10-25% of the urban adult population have chronic bronchitis, country dwellers have a lower incidence. Quite frequently chronic bronchitis is associated with emphysema.

The two most important etiologic factors responsible for the genesis of chronic bronchitis are chronic irritation by inhaled substances and microbiologic infections. Cigarette

smoking remains the paramount influence. Chronic bronchitis is 4-10 times more common in heavy smokers irrespective of age, sex , occupation and place of dwelling.

Grossly ; the bronchial wall is thickened, hyperaemic and edematous. Lumen of the bronchi and bronchioles may contain mucus plugs and purulent exudate.

Microscopically ; the cartilage containing large airways have hypertrophy and hyperplasia of submucosal glands. The bronchial epithelium may show squamous metaplasia and dysplasia. The non-cartilage containing small airways show goblet cell hyperplasia and intraluminal and peribronchial fibrosis.

2. Emphysema :

It is characterized by abnormal permanent enlargement of the air spaces distal to the terminal bronchiole accompanied by destruction of their walls.It is classified into 5 types according to the acinus involved, centriacinar (Centrilobular), Panacinar (Pan lobular), Paraseptal (distalacinar), Irregular (Para-cicatricial) and mixed (unclassified) emphysema.

Thurlbeck reports a 50% combined incidence of panacinar and centriacinar emphysema at autopsy. He considers the pulmonary disease to be responsible for the death of 6.5% of these patients.

The association of the chronic bronchitis and emphysema is principally linked to the tobacco smoke and air pollutants. In emphysema, the destruction of the alveolar walls, is not linked to bronchial changes but is closely related to deficiency of serum alpha-1-antitrypsin (alpha-1-protease inhibitor) commonly termed protease antiprotease hypothesis.

(i) Centriacinar Emphysema

It is one of the common types characterized by the invalvement of the central or proximal parts of the acini, formed by respiratory bronchioles sparing distal alveoli. It usually coexists with chronic bronchitis and occurs psedominantly in smokers and in coal miners pneumoconiosis.

Grossly ; the lesions are more common and more severe in the upper lobes of the lungs. Cut surface shows distended air spaces in the centre of the lobules surrounded by a rim of normal lung parenchyma in the same lobule. The lobule are separated from each other by fine fibrous tissue septa. Large amount of black pigment is often present in the walls of emphysematous spaces. In more severe cases, distal parts of acini are also involved and appearance may closely resemble panacinar emphysema.

Microscopically ; distention and destruction of the respiratory bronchiole in the centre of lobules, surrounded peripherally by normal involved alveoli. The terminal bronchioles

supplying the acini show chronic inflammation and are narrowed.

(ii) Panacinar (Pan lobular) emphysema -

In this type, all portion of the acinus are affected but not of the entire lung. Panacinar emphysema is most often associated with Alpha-1 - antitrypsin deficiency in middle aged smokers, produces the most characteristic anatomical changes in the lung in emphysema.

Grossly ; lower zone of lungs more frequently and more severely involved than the upper zone, The involvement may be confined to a few lobules, or may be more widespread affecting a lobe or part of a lobe of the lung. The lungs are enlarged and over inflated.

Microscopically ; usually all the alveoli within a lobule are affected to the same degree. All portions of acini are distended - respiratory bronchioles, alveolar ducts and alveoli , are all dilated and their walls stretched and thin. Ruptured alveolar walls and spurs of broken septa are seen between the adjascent alveoli. The capillaries are stretched and thinned. Special stains show loss of elastic tissue. Inflammatory changes are usually absent.

(iii) Paraseptal (Distal Acinar) Emphysema -

It involves distal part of acinus with normal proximal part. It is localized along the pleura and along the perilobular septa. It occurs adjascent to areas of fibrosis, scarring or

atelectasis and is usually more severe in the upper half of the lungs. It is the common cause of spontaneous pneumothorax in young adults. Grossly, the subpleural portion of the lung shows air-filled cysts, 0.5 to 2cm. in diameter.

(iv) Irregular (Para-cicatricial) Emphysema -

This is the most common form of emphysema, seen surrounding scars from any cause. The involvement is irregular as regards the portion of acinus involved as well as within the lung as a whole. During life irregular emphysema is often asymptomatic and may be only an incidental autopsy finding.

(v) Mixed (unclassified) Emphysema -

Quite often , the same lung may show more than one type of emphysema. It is usually due to more severe involvement resulting in loss of clear cut distinction between one type of emphysema and the other. Thus , the lungs of an elderly smoker at autopsy may show continuation of centriacinar emphysema in the upper lobes, panacinar in the lower lobes, and paraseptal emphysema in the subpleural region.

3. Bronchial Asthma -

It is characterized by increased responsiveness of the tracheobronchial tree to various stimuli, potentiating paroxysmal constriction of the bronchial air ways.

It is common and prevalent world wide , in USA its incidence is 4%. It occurs at all ages but nearly 50% of cases develop it before age of 10 years.

Asthma has traditionally been divided into two basic types : extrinsic (allergic, reagin mediated,atopic) and intrinsic (idiosyncratic, non-atopic) asthma, a third type is a mixed pattern of both.

Grossly ; the lungs are over distended due to over inflation. The cut surface shows characteristic occlusion of the bronchi and bronchioles by viscid mucus plugs.

Microscopically ; the mucus plugs contain curschmann's spirals, charcot leyden crystals, the bronchial wall shows thickened basement membrane of the bronchial epithelium. submucosal edema and inflammatory exudate consisting of lymphocytes and plasma cells with prominence of eosinophils. There is hypertrophy of submucosal glands and bronchial smooth muscles. Changes of bronchitis and emphysema may supervene, especially in intrinsic asthma.

4. Bronchiectasis -

It is a chronic necrotizing infection of the bronchi and bronchioles leading to or associated with abnormal and irreversible dilatation of these air ways ; manifested clinically by cough, fever and the expectoration of the copious amounts of foul-smelling, purulent sputum. The origin of inflammatory

destructive process of bronchial walls is nearly always a result of two basic mechanisms : obstruction and infection.

Grossly ; the lungs may be invalved diffusely or segmentally. Bilateral involvement of lower lobes occurs most frequently. More vertical air passages of left lower lobe are more often involved than the right. The pleura is usually fibrotic and thickened with adhesions to the chest wall. Cut surface of the affected lobes, generally the lower zones, shows characteristic honey-combed appearance. The bronchi are extensively dilated nearly to the pleura, their walls are thickened and the lumen are filled with mucus or muco-pus. The intervening lung parenchyma is reduced and fibrotic.

Microscopically ; In fully developed cases, the bronchial epithelium may be normal, ulcerated or may show squamous metaplasia. The bronchial wall shows infiltration by acute and chronic inflammatory cells and destruction of normal muscle and elastic tissue with replacement by fibrosis. The intervening lung parenchyma shows fibrosis, while the surrounding lung tissue shows changes of interstitial pneumonia. The pleura in the affected area is adherent and shows bands of fibrous tissue between the bronchus and the pleura.

IV. Chronic Restrictive (Diffuse Interstitial, Infiltrative) Pulmonary Diseases :

Characterized by reduced expansion of lung parenchyma with decreased total lung capacity. The condition included

in this group are : Pneumoconiosis, Immunologic lung diseases, Collagen - vascular diseases, Idiopathic pulmonary fibrosis and sarcoidosis. These disorders account for about 15% of non infectious disease seen by pulmonary physicians.

A. Pneumoconiosis :

Pneumoconiosis is the term used for lung diseases caused by inhalation of dust, mostly at work, therefore also called dust diseases or "occupational lung diseases".

The development of a pneumoconiosis is dependent on : the amount of dust retained in the lung and airways, the size and shape of particles, their solubility and physiochemical reactivity, the additional effect of other irritants such as tobacco smoke and host factors such as efficiency of clearance mechanism and immune status of the host.

1. Coal Worker s' Pneumoconiosis -

This is the commonest form of pneumoconiosis, defined as the lung disease resulting from inhalation of coal dust particles specially in coal miners engaged in handling soft bituminous coal for many years, often 20-30 years. It exists in two forms : milder form -simple coal workers' pneumoconiosis and advanced form - progressive massive fibrosis (complicated coal miner's pneumoconiosis).

Anthracosis is the common , benign and asymptomatic accumulation of carbon dust in the lung of most urban dwellers due to atmospheric pollution and cigarette smoke.

(i) Simple coal worker's Pneumoconiosis -

Grossly ; the lung parenchyma shows small black focal lesions, more in upper lobes in the form of coal macules or nodules.

Microscopically ; coal macules are composed of aggregates of dust laden macrophages, present in the alveoli and in the bronchiolar and alveolar walls ; increase in the network of reticulin and collagen in the coal macules. Respiratory bronchioles and alveoli surrounding the macules are distended without significant destruction of the alveolar walls.

(ii) Progressive massive fibrosis -

Grossly ; coal macules and nodules, larger hard, black scattered areas, usually bilateral and located in the upper parts of the lungs posteriorly. The pleura and regional lymph nodes are also blackened and fibrotic.

Microscopically ; the fibrous lesions are composed almost entirely of dense collagen and carbon pigment. The wall of respiratory bronchioles and pulmonary vessels included in the massive scars are thickened with obliterated lumen, the alveoli surrounding the scars are markedly dilated.

(iii) Rheumatoid pneumoconiosis (Caplan's syndrome) :

The development of rheumatoid arthritis in a few cases of coal workers pneumoconiosis, silicosis or asbestosis is termed rheumatoid pneumoconiosis or Caplan's syndrome. The lung lesion in caplan's syndrome have immunological

basis for their origin as evidenced by detection of rheumatoid factor and antinuclear antibodies.

Grossly ; the lungs have rounded , firm, nodules with central necrosis, cavitation or calcification.

Microscopically ; the lung lesions are modified rheumatoid nodules with central zone of dust- laden fibrinoid necrosis enclosed by palisading fibroblasts and mononuclear cells.

2. Silicosis -

Silicosis is caused by prolonged inhalation of silicon dioxide (silica). An infrequent acute form of silicosis called accelerated silicosis produces irregular fibrosis adjoining the alveoli which is filled with lipoproteinaceous exudate and resembles alveolar proteinosis.

Grossly ; the chronic silicotic lung is studded with well circumscribed, hard fibrotic nodules , scattered throughout the lung parenchyma, more often located in the upper zones, having coal dust deposition and calcification. Pleura is thickened and adherent to the chest wall.

Microscopically ; the silicotic nodules are located in the region of respiratory bronchioles, adjascent alveoli, pulmonary arteries, in the pleura and the regional lymph nodes. The silicotic nodules consist of central hyalinised material with scanty cellularity and some amount of dust. The collagenous nodule have cleft like, spaces between the lamellae of

collagen. The intervening lung parenchyma may show hyperinflation or emphysema.

3. *Asbetosis and asbestos - related lesions :*

Inhalation of asbestos causes asbestosis , pleural disease and tumors.

(i) Asbestosis

Grossly ; the affected lung are small, firm with cartilage like thickening of the pleura. The sectioned surface shows variable degree of pulmonary fibrosis especially in the subpleural areas and in the bases of lungs. The advanced cases may shows cystic changes.

Microscopically ; Non specific interstitial fibrosis, asbestos bodies in the invalved areas, there may be changes of emphysema in the pulmonary parenchyma between the areas of interstitial fibrosis.

(ii) Pleural disease :

Pleural disease in asbestos exposure may produce pleural effusion, visceral pleural fibrosis or pleural plaques.

Grossly ; the lesions appear as circumscribed , flat, small, firm or hard, bilateral nodules, often seen on the posterolateral part of parietal pleura and on the pleural surface of the diaphragm.

Microscopically ; they consist of hyalinised collagenous tissue which may be calcified so that they are visible on chest X-ray. Asbestos bodies are generally not found within the plaques.

(iii) Tumors -

Asbestos exposure predisposes to a number of cancers most importantly bronchogenic carcinoma and malignant mesothelioma. A few others are : carcinoma of esophagus, stomach, colon , kidneys, larynx and various lymphoid malignancies.

4. Berylliosis

It is caused by heavy exposure to dust or fumes of metallic beryllium or its salts. Two forms of pulmonary berylliosis-acute and chronic.

i) Acute Berylliosis

The pulmonary reaction is in the form of an exudative chemical pneumonitis in which alveoli are filled with protein rich fluid with formation of hyaline membrane.

ii) Chronic Berylliosis -

The condition is characterized by development of non-caseating epitheloid granulomas like those of sarcoidosis, diffusely scattered throughout the lung parenchyma.

B. Immunologic Lung Diseases :

Immunologic mechanisms play an important role in a number of lung diseases which includes ; Bronchial asthma,

Hypersensitivity(Allergic)Pneumonitis,Pulmonary eosinophilia, Good pasture's syndrome and pulmonary alveolar proteinosis.

(i). Hypersensitivity (Allergic) Pneumonitis

It is a group of immunologically mediated interstitial lung diseases occurring in workers inhaling a variety of organic (biologic) antigenic materials. The condition may have an acute onset due to isolated exposure or may be chronic due to repeated low dose exposure. It occurs due to various conditions like farmers lung , Bagassosis, Byssinosis, Bird-breeders' (Bird fanciers') lung, mushroom workers' lung, malt workers' lung, maple bark disease and silo-fillers' disease.

(ii) Pulmonary Eosinoiphilia-

Pulmonary eosinophilia, eosinophilic pneumonias or pulmonary infiltration with eosinophilia (PIE) syndrome are a group of immunologically mediated lung diseases characterized by combination of two features : infiltration of the lung in chest radiographs and elevated eosinophil count in peripheral blood. PIE syndrome has a number of diverse causes and pathogenesis which includes : Loffler's syndrome, Tropical pulmonary eosinophilia, secondary chronic pulmonary eosinophilia, Idiopathic chronic eosinophilic pneumonia and hypereosinophilic syndrome.

Grossly ; the lungs usually show patchy consolidation.

Microscopically ; thickening of the alveolar walls by edema and exudate, chiefly of eosinophils and some lymphocytes and plasma cells. The alveolar lumen also contain eosinophils.

(iii) Goodpasture's syndrome -

It is a combination of necrotizing hemorrhagic interstitial pneumonitis and rapidly progressive glomerulonephritis.

Grossly ; the lungs are heavy with red brown areas of consolidation.

Microscopically ; In acute stage - focal areas of hemorrhages in the alveoli and focal necrosis in the alveolar walls. In more chronic stages, organisation of the hemorrhage leading to interstitial fibrosis and filling of alveoli with hemosiderin - laden macrophages.

(iv) Pulmonary Alveolar Proteinosis-

It is a rare chronic disease in which the distal airspaces of the lungs are filled with granular , PAS positive eosinophilic material with abundant lipid in it. The condition can occur at any age from infancy to old age.

Grossly ; usually both lungs involved , particularly lower lobes, lungs are heavier with areas of consolidation, sectioned surface exudes abundant turbid fluid.

Microscopically ; presence of homogenous , granular eosinophilic PAS positive material ; often contain cholesterol clefts.

C. *Collagen vascular disease -*

A number of collagen diseases may result in chronic interstitial fibrosis and destruction of blood vessels ; includes scleroderma (progressive systemic sclerosis), Rheumatoid Arthritis, SLE , Sjogren's syndrome, Dermatomyositis and Polymyositis and Wegner's granulomatosis.

Microscopically ; these granulomas have foci of fibrinoid necrosis and intense exudate of lymphocytes, plasma cells and macrophages with scattered multinucleate giant cells. Besides necrotising granulomas, there is associated vasculitis.

D. *Idiopathic Pulmonary Fibrosis -*

It refers to a poorly understood pulmonary disorder characterized histologically by diffuse interstitial and inflammation fibrosis, which in the advanced cases results in severe hypoxaemia and cyanosis. The pathogenesis of idiopathic pulmonary fibrosis is unknown and the condition is diagnosed by excluding all known causes of interstitial fibrosis.

Grossly ; the lungs are firm, heavier with reduced volume, Honey - combing may be present in the subpleural region.

Microscopically ; In early stage - widening of alveolar septa, hyperplasia of alveolar lining cells at places , often hyaline membrane formation, In advanced stage, organisation of the alveolar exudate, replacement fibrosis in the alveoli and interstitial septal wall with variable amount of inflammation.

V. Tumors of Lungs :

A variety of benign and malignant tumors may arise in the lung but the vast majority (90-95%) are bronchogenic carcinomas, 5% are bronchial carcinoids and 2-5% are mesenchymal and other miscellaneous neoplasms.

A Bronchogenic carcinoma -

It is the common visceral malignancy in males accounts for about one third of all cancer death in males and one twentieth of all deaths in both sexes. The high incidence of lung cancer is associated with a number of etiologic factors, most important of which is cigarette smoking. About 80% of lung cancer occurs in active smokers. Other etiologic factors are atmospheric pollution, occupational causes, dietary factors, genetic factors and chronic scarring.

Bronchogenic carcinoma can occur anywhere in the lung but the most common location is hilar followed by peripheral type.

Grossly , two main types show variation in appearance :

(i) *Hilar Type* - Thickened bronchial mucosa producing nodular or ulcerated surface. Cut surface of the tumor is yellowish white with foci of necrosis and homogenous, may produce cavitary lesion.

(ii) *Peripheral Type* - Single or multiple nodules in the periphery of the lung producing pneumonia like

consolidation of a large part of lung. The cut surface of the tumor is grayish & mucoid.

Microscopically : divided into five types.

1. *Squamous cell (epidermoid) carcinoma.*

This is the most common type (35-50%) of bronchogenic carcinoma, more common in men , particularly with history of tobacco smoking. These tumors usually arise in large bronchus and prone to massive necrosis and cavitation. The tumor is diagnosed microscopically by identification of either inter cellular bridges or keratinisation. The tumor may show varying histologic grades of differentiation such as well differentiated, moderately differentiated and poorly differentiated.

2. *Small cell carcinoma (20-25%)*

Small cell carcinomas are frequently hilar or central in location, have strong relationship to cigarette smoking and are highly malignant tumors. They are most often associated with ectopic hormone production because of the presence of neurosecretory granules in majority of tumor cells which are similar to those found in argentaffin or kulchitsky cells normally found in bronchial epithelium. Small cell carcinoma have three subtypes.

(i) *Oat cell carcinoma* - is composed of uniform, small cells larger than lymphocytes with dense, round or oval nuclei having diffuse chromatin, inconspicuous nucleoli and

very sparse cytoplasm. These cells are organized into cords aggregates and ribbons or around small blood vessels forming pseudorosettes.

(ii). *Small cell carcinoma* - intermediate cell type is composed of cells slightly larger than those of oat cell carcinoma and have similar nuclear characteristics but have more abundant cytoplasm. These cells are organized into lobules.

(iii) Combined oat carcinoma is a tumor, having definite component of oat cell carcinoma with squamous cell and or adenocarcinoma.

3. *Adenocarcinoma (15-35%)*

Adenocarcinoma, also called peripheral carcinoma due to its location and scar carcinoma due to its association with areas of chronic scaring , is the most common bronchogenic carcinoma in women and is slow growing. It is further subclassified into four types :

(i) Acinar adenocarcinoma has predominance of glandular structure and often occurs in larger bronchi.

(ii) Papillary adenocarcinoma has a pronounced papillary configuration and is frequently peripherally located in the lungs and is found in relation to pulmonay scars (Scar carcinoma).

(iii) Bronchiolo - alveolar carcinoma is characterized by cuboidal cells growing along the existing alveoli and forming numerous papillary structures.

Ultra structurally , these tumor cells resemble clara cells or less often type II pneumocytes.

(iv) Solid carcinoma is a poorly differentiated adeno-carcinoma lacking acini, tubules or papillae but having mucus containing vacuoles in many tumor cells.

4. *Large cell carcinoma (10-15%)*

These are undifferentiated carcinomas which lack the specific features by which they could be assigned into squamous cell carcinoma or adenocarcinoma. Large cell carcinomas are more common in men, have strong association with cigarette smoking and are highly malignant tumors. The tumor cells have large nuclei , prominent nucleoli abundant cytoplasm and well defined cell borders. Variants of large cell undifferentiated carcinomas include giant cell carcinoma with prominence of highly pleomorphic multinucleate cells and clear cell carcinoma composed of cells with clear or foamy cytoplasms without mucin.

5. *Adenosquamous Carcinoma (1-3%)*

These are a small proportion of peripheral scar carcinomas having clear evidence of both keratinisation and glandular differentiation.

B. Bronchial Carcinoids

Bronchial carcinoids are tumors of low grade malignancy arising from neuroendocrine (kulchitsky) cells of bronchial mucosa.

Grossly ; spherical polypoid mass , intact bronchial mucosa, cut surface of tumor is yellow-tan in colour.

Microscopically ; tumor is composed of uniform cuboidal cells forming aggregates , trabeculae or ribbons separated by fine fibrous septa.

C. Metastatic lung tumors

Secondary tumor of the lungs are more common than the primary pulmonary tumors. Metastasis are most common in the peripheral part of the lung forming single or multiple , discrete, nodular lesion which appear radiologically as "Cannon-ball secondaries".

VI. Pleural Tumors

Pleural tumors may be primary or secondary. The secondary tumors in the pleura are more common. The only important primary tumor of pleura is mesothelioma.

1. Mesothelioma

It is an uncommon tumor arising from mesothelial lining of serous cavities , most often in pleural cavity. It is of two types.

i) Benign (Solitary) Mesothelioma (Pleural fibroma) -

Grossly ; solitary, circumscribed, small, firm mass. Cut surface shows whorls of dense fibrous tissue.

Microscopically ; Predominantly composed of whorls of collagen fibres and reticulin with interspered fibroblasts.

ii) Malignant (Diffuse) Mesothelioma -

About 90% of malignant mesotheliomas are asbestos related.

Grossly : diffuse , forming a thick, white , fleshy , coating over the parietal and visceral surfaces.

Microscopically : malignant mesothelioma may have epithelial, sarcomatoid or biphasic patterns. Asbestos bodies are found in the lungs of most patients with malignant mesothelioma of any histologic type.

Historical Aspect of Autopsy -

In ancient times human dissection were known to have been advocated as early as 1000 B.C. (Anderson,1996).

In medieval period appreciation for the human form and the details of anatomy would await the great anatomist Andreas Vesalius.

Other works in renaisance period brought new life to the autopsy. Benivieni's publications in1507 presented information on 110 patients, out of which autopsy was performed on 15 cases. Theophilus Bonetus's monumental work in 1679 included description of 3000 autopsies. There

after Hermann Boerhaave (1668-1738) wrote two monographs dealing with autopsy methodology and Geovanni Battista Morgagni (1682-1771)popularized the fledging activity of clinicopathological correlation.

In pre-modern period Marie Francis Xavier Bichat (1771-1802) provided fresh intellecutual and compassionate ingradients common to the 18th century. Jean Nicholas corvisart des Marets (1755-1821) made extensive contribution to our under standing of cardio-vascular diseases. Theophile Hyacinthe Laennec work included description of the clinical and pathologic features of tuberculosis, further illustrated commitment to an anatomic basis of medical care which was fortified by the renowned contemporaries in pathology Carl Von Rokitansky (1804-1878) and Rudolf Virchow (1821-1905).

In 20th century, influence of physician in the mold of Bichat and Sir william Osler was represented in text books of medicine, in clinicopathological conferences. The concept of quality assurance through the autopsy was emerging. The land mark survey of medical education programs across the united State and Canada by Abraham Flexner in 1910 had lead to many recommendations and reform.

Different Autopsy Studies -

Autopsy studies for pulmonary diseases have been conducted by various workers viz.

Moon (1948) carried out autopsy study for shock lung and found that lungs were heavy and showed patchy alteration of varying extent and irregular distribution. The affected portions were dark, plum coloured and air less.

Willis (1953), Olcut (1956) studied Lung tumor heterogeneity for over 30 years and further emphasized by Roggli et al (1985). They examined 100 consecutive lung carcinoma, a mixture of resection and autopsy material and reviewed the whole tumor or a minimum of 10 blocks of tissue from each.

Scully and Captain in 1956 have reported 30.15% of severe grade of pulmonary embolism.

Peltier (1957) recorded higher incidence of severe grade of pulmonary fat embolism in simple than compound fracture cases.

Anderson et al (1962) studied that the longer the duration of labour and longer the time after rupture of membranes, more are the chances of developing intrauterine pneumonias.

Butler and Bonham (1963) published in their article based on the 1958 British perinatal mortality survey that at autopsy pneumonia was present in 0.15%, Hyalinemembrane in 0.16%, and massive pulmonary haemorrhage in 0.06% total births. The datas were compared with those published in

British Birth Survey 1970 (Claireaux, 1975) that at autopsy pneumonia was present in 0.01%, Hyaline membrane in 0.31% and massive pulmonary hemorrhage in 0.02% total births. It was also compared with records of perinatal deaths among babies born at Queen charlotte's Maternity hospital between 1963 and 1977 in three 5 year period which showed between 1963-67, 1968-72, 1973-77, pneumonia was present in 0.04%, 0.06% and 0.02% total births respectively, Hyaline membrane was present in 0.19%, 0.18% and 0.09% total births respectively and massive pulmonary hemorrhage was present in 0.06%, 0.05% and 0.02% total births respectively.

Palmovic and Mc Caroll (1965) have reported 44% of severe grade of pulmonary embolism in trauma cases.

Wahal et al (1967) studied pathology of lung in 123 cases of perinatal death and found that pneumonia was a rare cause of fetal death.

Teplitz (1968) performed autopsy for shock lung. Lungs were heavy and showed patchy alteration of varying extent and irregular distribution. The affected portion were dark, plum coloured and airless.

Kim et al (1976) performed 768 consecutive autopsies between 1972 and 1974, out of which 24 cases were histologically verified DIC, fibrin thrombi were present in 13 cases of lung.

Thurlbeck (1976) reported a 50% combined incidence of panacinar and centriacinar emphysema at autopsy.

Valdivia et al (1977) studied lung specimens in 30 cases including 3 autopsies, with a clinical diagnosis of Idiopathic interstitial pneumonitis, six cases had intra alveolar lesion believed to be early, while 20 had advanced disease characterized by intra alveolar cellular clumps, alveolar wall fibrosis, distortion and loss of pulmonary parenchyma.

Cechner et al (1980) studied series of 415 deaths in which clinically occult bronchogenic carcinoma was discovered in 28% cases at autopsy.

Sharma et al (1980) studied a case of Ebstein disease by autopsy in which lungs showed firm, fibrous, pleural adhesions on both side. Lungs weighed 145 gm each and were riddled with multiple firm nodules 1-3mm in diameter, some having central areas of caseation. Microscopically both lungs showed multiple caseating tuberculous granulomas with involvement of the hilar lymphnodes.

Yesner & carter (1982) studied histological classification and tumor behaviour in which well and moderately differentiated squamous carcinoma are confined to the thorax in 60% cases at autopsy.

Patil et al (1983) studied 301 neonatal autopsies performed during 1979 and 1981, 190 autopsies (63.1%)

revealed lung as a primary cause of death, out of which 58 cases were identified to be Hyaline membrane disease (H.M.D.).

Agarawal et al (1983) studied that the incidence of pulmonary and renal fat embolism was 92% and 61.4% respectively in autopsy material from road accidents cases by osmic staining. Incidence of pulmonary edema (66.4%) and the renal fat embolism was directly proportional to the grade of pulmonary fat embolism.

Naik et al (1987) studied fifty cases of fetal death between June 1980 and Dec. 1981, with clinicopathological correlation. It was found that anoxia was the major cause of fetal death (72%) and pneumonia was cause of death in 2% cases.

In compilation by *Nemetz et al (1987)* and *Wheeler (1987),* the possibility of a missed clinical diagnosis ranged from as little as 4% to as much as 66% verified at autopsy.

Mac Gee W (1993) studied 3000 consecutive autopsies performed between 1972 and 1992 in geriatric institution aged between 62-102 years, found that the most common fatal conditions were acute infections mainly bronchopneumonia (42.9%) and urinary tract infection (12.3%), malignant neoplasm particularly of gastointestinal tract and its adnexae and the lungs (28.1%), pulmonary

thromboembolism (21.2%) and acute myocardial infarction (19.6%).

Czegledy et al (1993) studied 881 autopsies of infants between 1987 and 1990, found that most often pleural petechae were combined with petechial haemorrhages in the thymus and epicardium.

Helweg et al (1993) reviewed 68 autopsy findings and histologic sections, revealed no cause of death in 11 of the 27 natural deaths. Seven of those deaths had previously ascribed to infectious disease or aspiration and 3 had been registered as uncertain cause of death.

Szende et al (1994) compared pre and post autopsy diagnosis in 2000 consecutive autopsies in individuals aged 30-80 years. At autopsy the underlying cause of death due to respiratory system was 2.2%.

Klatt et al (1994) studied 565 autopsies in adults with AIDS between 1982 and 1993 in which for all patients, Pneumocystis carinii pneumonia (PCP) was the most common AIDS related cause of death during the entire period evaluated, followed in frequency by bacterial sepsis, fungal infections, malignant lymphoma, cytomyalovirus (CMV) infections, tuberculosis , Kaposi's sarcoma, toxoplasmosis, encephalopathy and mycobacterium avium complex infection.

Zaki et al (1995), studied 44 patients have died of Hantavirus pulmonary syndrome (HPS) since mid 1993 , All but 4 of the 44 patients who died exhibited interstitial pneumonitis with varying numbers of infiltrating mononuclear cells , edema and focal hyaline membrane formation. Hantaviral antigen were identified in the endothelium of microvessels particularly in the lungs.

Nolte et al (1995) confirmed Hantavirus infection by serology, polymerase chain reaction (PCR), identification of hantaviral gene sequence in autopsy tissue or immunohistochemical staining of autopsy tissue for hantravirus antigens. Of the 22 patients aged 12-68 years with Hantavirus pulmonary syndrome (HPS), 9 survived and 13 died.

Saller et al (1995) studied a total of 168 perinatal autopsies and concluded that perinatal autopsy was particularly beneficial in changing 29.9% of clinical diagnosis and confirming 28.9% of them.

Pandya et al (1996) studied 100 neonatal autopsies in which pulmonary pathology was major cause of death (79%) and among these pneumonia was the leading cause (48%) followd by Hyaline membrane disease (HMD) (13%), respiratory distress was noted in 41% caused by pneumonia (34%), HMD (31%), massive pulmonary haemorrhage (MPH) (17%), Pneumothorax (17%), and meconium aspiration 7%. Among pneumonias, bronchopneumonia was the major lesion

(50%) followed by interstitial pneumonia (14%), aspiration pneumonia (10%) and fungal pneumonia (8%). Orphans have higher susceptibility to pneumonia (71%) than non-orphans (41%). Pulmonary Haemorrhage was noted in 47% of cases, of which 13% showed massive haemorrhage. Patchy emphysema (83%) and patchy atelectasis (68%) were common features in the lungs of neonates.

Joste et al (1996) studied a case of Albendazole therapy for microsporidiosis, findings of which were verified by autopsy, in which the immediate cause of death was acute bacterial pneumonia and post mortem bacterial culture from lungs and sinuses were positive for Pseudomonas aeruginosa.

Moore et al (1996), published results of 1625 consecutive fetal and neonatal autopsy face sheets spanning 20 years between 1975 and 1994. The most common cause of death in group A (Likely immediate cause of death) was fetal pneumonia (13%). In group B (likely intermediate cause of death), the most common cause of death was Hyaline membrane disease (15%).

Feinstein (1996) and his collegues studied a consecutive series of 2996 (non medicolegal) autopsies between 1972 and 1981 of those 110 were ineligible of their calculations ; among the 2886 eligible necropsies primary lung cancer was first found at necropsy in 49 cases.

Haque et al (1996) observed at their institution clinically undiagnosed and unsuspected pulmonary thromboemboli and acute myocardial infarction, as well as sepsis, were found to be the most common cause of unexpected deaths found at autopsy in surgical patients.

Sehonanda et al (1996) analyzed 168 autopsies performed in AIDS patients between 1982 to 1993. The lungs were most commonly involved organ (80%).Through 1986 , 75% of AIDS autopsies demonstrated single infection i.e. Pnemocystis carinii pneumonia (PCP). Since 1987 , 72% of autopsies demonstrated multiple infections related to PCP , Mycobacteriosis , CMV and various fungi . During the last 3 years the prevalence of mycobacterial infection was higher than in the previous 9 years combined. In contrast PCP decreased from 52% in 1988 to 14% in 1993.

Hill and Anderson (1996) in their article showed the partial list of diseases discovered or critically clarified through autopsy since 1950, which includes bronchopulmonary lesions such as Alveolitis (difuse alveolar damage, shock lung, respiratory distress syndrome), oxygen toxicity, Pneumocystis pneumonia , Infantile respiratory distress syndrome (HMD), Legionnaire's disease, pulmonary alveolar proteinosis, desquamative pneumonia, diseases due to inhalation of industrial dusts (Asbestosis, berylliosis, bagassosis, silio-filler's disease), Lipid pneumonia, diffuse interstitial fibrosis.

Madyastha (1996), reported a case of malignant osteoporosis which clinically developed features of ARDS, Which was confirmed on autopsy.

Akikusha et al (1997) studied 25 autopsy cases of microscopic polyangiitis, showed necrotizing alveolar capillaritis in 40% cases.

Onadeko et al (1997), studied clinicopathologic pattern of carcinoma of the bronchus and lung over a 20 years period 1971-1990 by clinical , histopathological and autopsy study. The findings suggested that increasing industrialization and increased acquisition of modern diagnostic facilities are responsible for the increased incidence.

Saleh et al (1997) reported a case of lung carcinoma initially presented as a submandubular gland swelling diagnosed by aspiration biopsy.

Necropsy studies shown that pulmonary embolism contributes to another 5-10% of deaths occuring outside hospital. Mortality rate of an untreated case of pulmonary embolism is around 30% (Mittal et al, 1998).

In a retrospective autopsy study of *Bhatia et al (1998)* from January 1982 to December 1992 , total 10037 autopsies were performed in which 0.87% patients dying of bleeding diathesis out of which lungs were involved in 20 cases.

Shet et al (1998) studied 14 paediatric autopsies of neonatal hepatitis out of 1463pediatric autopsies between 1986 and 1944 in which interstitial pneumonia was seen in five cases.

An autopsy study was carried out by Lanjewar et al (1998) of 92 AIDS patients in India , between 1989 and 1996 in which lungs were invalved in 71 cases. 64 cases have shown tuberculosis, Cytomegalovirus infection in 3 cases and Cryptococcus neoformans infection in 4 cases.

Vaideeswar P. (1998) reported a case of resolving myocarditis and dilated cardiomyopathy who developed spontaneous right sided pneumothorax. Autopsy revealed rupture of cavitary pulmonary infarction to be the cause of the pneumothorax, a rare finding.

Chopra et al (1999) reported a case of Acute pneumonitis with pulmonary haemorrhage an uncommon and potentially fatal complication of SLE, in which autopsy was performed.

Lanjewar et al (1999) represented the first complete autopsy report of Pneumocytis carinii pneumonia in patients with AIDS from India. On autopsy the examination of both the lungs showed normal pleural surface, as well as cut surface. On histopathological examination foamy eosinophilic intraalveolar exudate in lung indicative of Pneumocytis carinii pneumonia (PCP).

Remiszewski et al (1999) studied frequency of pulmonary thromboembolism in small cell lung cancer (SCLC) patients treated in the institute of TB & chest diseases in the years 1980-1994. The frequency of pulmonary thromboembolism was 8% at autopsy.

Asano et al (1999) reported a rare case of sudden death of a patient with acute pulmoanry thromboembolism associated with chlorine gas poisoning. An autopsy revealed bilateral pulmonary thromboembolism.

Tomioka et al (1999) studied an autopsy case of interstitial pneumonia probably induced by sho-saiko-to, showed diffused alveolar damage and honey combing at autopsy. Furthermore, reverse transcriptase polymeraze chain reaction techniques detected HCV-RNA in specimen of fibrotic lung tissue.

Meir et al (1999) studied a case of complicated pulmonary tuberculosis, autopsy of which confirmed the cause of death due to fulminant pulmonary tuberculosis.

Hutchins et al (1999) studied a case of sudden death in a child due to an intrathoracic paraganglioma. Autopsy revealed a large posterior mediastinal mass that completely compressed the upper lobe of the right lung and the associated airways. The histologic and immunocytochemical features were that of a paraganglioma.

Muzuch (2000) treated a case of primary oesophageal carcinoma by transhiatal oesophagectomy without thoracotomy with a survival period of over 6 years with no relapse.

Sakai et al (2000) studied clinical features of 25 patients with Cytomegalovirus infection complicating hematological diseases unrelated to allogenic bone marrow transplantation. Nine cases were diagnosed by histopathology and 5 of these 9 cases were discovered as having CMV infection at autopsy.

Inayama et al (2000) reported a fatal case of death due to unusual aspiration of sardine fry in an elderly Japanese man with lung cancer. Autopsy revealed peculiar materials with all nests and pigmented particles, together with striated muscle and skin in the ectatic bronchioles of the left lower lobe.

Maeda et al (2000) reported a case of serial homicide by injection of succinylcholine by autopsy examination of five victims burried in a rural area, of which two victims showing moderate decomposition (about 3 months after death), intense pulmonary edema with pleural effusion.

Weng et al (2000) reported a case of multilocular thyroid gland ectopy, the autopsy revealed ectopic thyroid tissue in the lung, as a tumor mass under the visceral pleura.

Somaschini et al (2000) concluded in their study that diagnosis of misalignment of pulmonary vessels (MPV) and

alveolar capillary dysplasia should be considered during autopsy of infants who have died of irreversible persistent pulmonary hypertension.

Havlik et al (2000) reported 8 cases of pulmonary capillary hamangiomatosis (PCH) like foci that were incidental findings at autopsy in which the patients did not have symptoms of pulmonary hypertension nor did PCH contribute in any way to death.

Bubendorf et al (2000) studied 19,316 routine autopsies performed from 1967 to 1995 on men older than 40 years of age, the reports from 1,589 (8.2%) with prostate cancer were analyzed. Haematogenous metastasis were present in 35% of 1,589 patients with prostate cancer showed lung involvement in 46% cases and pleura in 21% cases.

Kurkciyan et al (2000) concluded in their study that pulmonary embolism is a possible non-cardiac cause of cardiac arrest. Mortality is very high and often diagnosis is established only by autopsy. In a retrospective study within 8 years, pulmonary embolism was found as the cause in 60 (4.8%) of 1246 cardiac arrest victims, in which 18 patients (30%) the diagnosis of pulmonary embolism was established only by postmortem.

Paloyan et al (2000) tried lung transplantation in a case of localized Bronchiolo alveolar carcinoma (BAC). The patient

expired 16 months after transplantation of other causes. The autopsy showed no evidence of recurrent BAC in the lungs or of metastatic lesion at any site.

Wick et al (2000) described two cases who experienced fatal rupture of pulmonary infarcts.

In a retrospective cohort study of *Costello et al (2000)* from 1977 to 1998, of 78 women with definite Tuberous selerosis complex (TSC), had evidence of pulmonary lymphangioleiomyomatosis (LAM) , surgical lung biopsy or autopsy in 7 patients confirmed the diagnosis of their lung disease.

Kameyama et al (2000) reported two autopsy cases of carcinoma that metastasized to a thyroid follicular adenoma. Autopsy of first case revealed that the metastatic colonic carcinoma was located in a thyroid follicular adenoma. Autopsy of second case revealed a metastatic lung adeno carcinoma also located in a follicular adenoma as in the first case. The phenomenon of tumor to tumor metastasis is rare.

Kodama et al (2000) studied a case of non tuberculous mycobacterial (NTM) infection followed for 12 years. Autopsy revealed cystic bronchiectasis accompanied by bronchial wall thickening in both lungs with some granuloma and acidfast bacteria observed in lung tissue.

Taura et al (2000) reported two cases of intravascular lymphomatosis diagnosed antemortem by transbronchial lung biopsy (TBLB).

Katsuoka et al (2000) reported an autopsy case of cerebral infarction with primary lung cancer. The autopsy findings revealed a primary papillary adenocarcinoma in the right lower lung, multiple cerebral infarction, renal infarction, pulmonary infarction and splenic infarction.

Russl et al (2000) studied a case of fatal fungal infection with complicating aortic dissection following coronary artery by pass grafting, autopsy of which revealed candida infection of the graft with a secondary aortobronchial fistula.

Franquet T et al (2000) reported semiinvasive pulmonary aspergillosis in COPD in nine patients in which semiinvasive aspergillosis had proven at autopsy in 7 cases. Aspergillosis colonies were identified within the lung tissue.

Totani et al (2000) studied a case of slicosis characterized by increasing serum CA 19-9 in parellel with progression of lung fibrosis, lung autopsy specimens disclosed severe interstitial fibrosis with prominent silicotic nodules.

Chan et al (2000) reported a case of sudden death from massive pulmonary embolism due to hepatocellular carcinoma. At autopsy the main pulmonary arteries of both

lungs were blocked by large tumor emboli , the immediate cause of death.

Tajima et al (2000) reported the first case of diffuse alveolar hemorrhage in an HTLV-I carrier.The autopsy revealed diffuse alveolar hemorrhage with no findings, of vasculitis.

Magee et al (2000) reported a case of acute pelvic thrombophlebitis missed by MRI of the pelvic veins.The woman presented post natally with pulmonary hypertension. Autopsy revealed right iliac vein thrombosis.

Takahashi et al (2000) studied a case of paraneoplastic pemphigus associated with bronchiolitis obliterans. Autopsy findings confirmed luminal narrowing of bonchioles by scarring which is a histopathologic feature of bronchiolitis obliterans.

Kaminsky et al (2000) reported a case of pulmonary leukostasis mimicking pulmonary embolism. Autopsy examination revealed extensive infiltration of leukaemic cells in all major organs with no evidence of pulmonary embolism.

Yoshida et al (2000) studied a case of Hemopneumothorax and hemoperitoneum in a case with large cell carcinoma of lung. The autopsy demonstrated hepatic metastasis and a ruptured adrenal metastasis.

Schweyer et al (2000) reported a case of malignant fibrous histiocytoma of the liver. Autopsy revealed a tumor measuring 8 cm in diameter which was located in the right lobe of liver and invaded the inferior vena cava. Because of multiple tumor aggregates seen in the left and right main pulmonary arteries, acute tumor embolisation of the lungs was regarded as cause of death.

Fujita et al (2000) reported a case of MFH of the lung by bronchial brushing cytology. An autopsy confirmed the diagnosis of MFH .

Nakamura et al (2000) studied a case of pulmonary adenocarcinoma with over expression of multidrugresistance associated protein and P53 aberration. The autopsy revealed pleural dissemination and intrapulmonary metastasis.

Herget et al (2000) studied a case of bursitis, spondylitis and aortic valvarendocarditis. Autopsy revealed as cause of death left heart failure with aortic valvar endocarditis and gelatinous pneumonia caused by late tubercular dessemination from the tubercular spondylitis.

OBSERVATIONS

OBSERVATIONS

The present study was conducted in the Department of pathology, M.L.B. Medical College & Hospital Jhansi, comprises analysis of total of 55 autopsies from the mortuary of M.L.B. Medical College & Hospital , Jhansi, also included one autopsy case from the military Base Hospital, Cantt. Jhansi.

Lung Tissue specimens were collected from bodies during postmortem in all cases and further subjected to histopathological examination to assess the incidence and pattern of different lung diseases in Bundelkhand region of U.P., Following observations were made :-

TABLE I

Sex Wise Distribution Of Cases (Victims) Autopsied

(Total cases - 55)

S.No.	Sex	No. of Victims	Percentage (%)
1.	Male	30	54.55
2.	Female	25	45.45

Table I shows sex wise distribution of victims studied. There were 30 male bodies (54.55%) and 25female bodies (45.45%).

TABLE II

Age Wise Distribution Of Cases Autopsied

(Total cases - 55)

S.No.	Age (in years)	No. of Cases	Percentage (%)
1.	11 - 20	8	14.55
2.	21 - 30	14	25.45
3.	31 - 40	16	29.09
4.	41 - 50	11	20.00
5.	51 - 60	4	7.27
6.	61 - 70	2	3.64

Table IV shows age wise distribution of cases autopsied. Maximum number of 16 cases(29.09%) were observed between 31-40 years age range, followed by 14 cases (25.45%)in 21-30 years age range, 11 cases (20.00%) in 41-50 years age range, 8 cases (14.55%) in 11-20 years age range, 4 cases (7.27%) in 51-60 years age range and 2 cases (3.64%) were in 61-70 years age range respectively.

TABLE III

Geographical area (Rural/Urban) wise distribution of cases autopsied

(Total cases - 55)

S.No.	Rural/Urban	No. of Cases	Percentage (%)
1.	Rural	32	58.18
2.	Urban	23	41.82

As evident from Table III, 32 cases (58.18%) belonged to rural areas and remaining 23 cases (41.82%) belonged to urban areas.

TABLE IV

Religion wise distribution of cases studied

(Total cases - 55)

S.No.	Religion	No. of Cases	Percentage (%)
1.	Hindu	47	85.45
2.	Muslim	6	10.91
3.	Sikh	2	3.64
4.	Others	-	-

Above Table shows religion wise distribution. Maximum number of cases were Hindus 47 (85.45%) whereas 6 cases (10.91%) were Muslims and 2 cases (3.64%) were Sikhs.

TABLE V

Time Since death

(Total cases - 55)

S.No.	Time Since death (in hours)	No. of Cases	Percentage (%)
1.	1 - 10	4	7.27
2.	11 - 20	47	85.46
3.	21 30	4	7.27

Table V shows time interval between death and the time at which autopsy/ postmortem was performed. Time since death was 11 - 20 hours in 47 cases (85.46%) and 1 - 10 hours and 21 - 30 hours in 4 cases (7.27%) each.

TABLE VI

Cause of death as ascertained among cases autopsied

(Total cases - 55)

S.No.	Cause of death	No. of cases	Percentage (%)
1.	Accidental (30)		(54.55)
	a) Road Traffic accidents	16	29.09
	b) Drowning	6	10.91
	c) Hanging	6	10.91
	d) Gunshot injuries	2	3.64
2.	Burn Injuries (14)		(25.45)
	a) Thermal burn	13	23.63
	b) Electric burn	1	1.82
	c) Others	-	-
3.	Poisoning (9)		(16.36)
	a) Organophoshorus	9	16.36
	b) Others	-	-
4.	Medical illness (2)		(3.64)
	a) Myocardial Infarction	1	1.82
	b) Unknown	1	1.82

As is evident in Table VI, in most of cases the cause of death was Accidental in 30 cases (54.54%) which included 16 cases (29.09%) of road traffic accidents, 6 cases (10.91%) of drowning and hanging each and 2 cases (3.64%) of gun shot injuries. Burn injuries encountered as cause of death in 14 cases (25.45%) which included 13 cases (23.63%) of thermal burn and one case (1.82%) of electric burn. In 9 cases (16.36%) cause of death was poisoning due to ingestion of organophosphorus compounds. In 2 cases (3.64%) death was due to medical illness which included one case (1.82%) of myocardial infarction and in one case (1.82%) the cause of death was not ascertained.

TABLE VII

Gross findings of lung specimens removed

(Total cases - 55)

S.No.	Cause of death	No. of cases	Percentage (%)
1.	Color		
	(i) Grayish white	19	34.54
	(ii) Grayish black	28	50.91
	(iii) Reddish brown	8	14.55
2.	Cut Surface		
	(i) Grayish white	19	34.54
	(ii) Grayish black	28	50.91
	(iii) Reddish brown	8	14.55
3.	Other findings		
	(i) Cysts	1	1.82
	(ii) Calcification	1	1.82

Table VII shows gross findings of lung specimens removed. The color and cut surface of lung specimens were grayish black in 28 cases (50.91%), grayish white in 19 cases (34.54%) and reddish brown in 8 cases (14.55%). Regarding other findings cysts and calcification were found in one case (1.82%) each.

TABLE VIII

Pathological findings / diagnosis observed in lung specimens studied

(Total cases - 55)

S.No.	Pathological findings / diagnosis	No. of cases	Percentage (%)
1.	Pulmonary Congestion	53	96.36
2.	Pulmonary edema	48	87.27
3.	Pneumonic consolidation	29	52.72
4.	Emphysema	19	34.54
5.	Chronic bronchitis	8	14.54
6.	Bronchopneumonia	7	12.73
7.	Tuberculosis	4	7.27
8.	Microthrombi	14	25.45
9.	Bronchiolar epithelial change	(14)	(25.45)
	(i) Hyperplastic change	12	21.81
	(ii) Metaplastic change	02	3.64
10.	Carbon Pigmentation	14	25.45
11.	Thickened alveolar septa	7	12.73
12.	Calcification of vessels	5	9.09
13.	Pulmonary alveolar fibrosis	4	7.27

Table VIII shows different pathological findings/ diagnosis as were observed in lung specimens removed for study.

Pulmonary congestion was present in 53 cases (96.36%) while pulmonary edema was present in 48 cases (87.27%). Pneumonic consolidation was found in 29 cases (52.72%). Emphysema was observed in 19 cases (34.54%) while chronic bronchits was found in 8 cases (14.54%). Bronchopneumonia was present in 7 cases (12.73%) and tuberculosis was found in 4 cases (7.27%). Microthrobi were observed in 14 cases (25.45%). Regarding bronchiolar epithelial change, it was present in 14 cases (25.45%) which included hyperplastic changes in 12 cases (21.81%) and metaplastic changes in 2 cases (3.64%). Increase carbon pigmentation was found in 14 cases (25.45%). Thickened alveolar septa was observed in 7 cases (12.73%). Calcification of vessels was present in 5 cases (9.09%), while pulmonary alveolar fibrosis was found in 4 cases (7.27%).

TABLE IX

Grading of Pulmonary congestion in cases studied

(Total cases - 53)

S.No.	Grading of Pulmonary Congestion	No. of cases	Percentage (%)
1.	Mild (+)	32	60.38
2.	Moderate (++ / +++)	13	24.53
3.	Severe (Heavy) (++++)	8	15.09

As evident from Table IX, shows arbitrary gradings of Pulmonary congestion. Mild Pulmonary congestion was present in 32 cases (60.38%) while moderate Pulmonary congestion was seen in 13 cases (24.53%). Heavy Pulmonary congestion was observed in 8 cases (15.09%).

TABLE X

Grading of pulmonary edema in cases studied

(Total cases - 48)

S.No.	Grading of Pulmonary Edema	No. of cases	Percentage (%)
1.	Mild (+)	27	56.25
2.	Moderate (++ / +++)	8	16.67
3.	Severe (++++)	13	27.08

Table X shows arbitrary grading of pulmonary edema in cases studied. Mild pulmonary edema was present in 27 cases (56.25%) and severe pulmonary edema was seen in 13 cases (27.08%) while moderate pulmonary edema was observed in 8 cases (16.67%).

TABLE XI

Geographical area wise distribution of Histopathological findings observed

Sl. No.	Histopathological findings	Total No. of cases (%)	Urban	Rural
			No. of cases (%)	No. of cases (%)
1.	Pulmonary Congestion	53 (96.36)	22 (41.51)	31 (58.49)
2.	Pulmonary edema	48 (87.27)	19 (39.58)	29 (60.42)
3.	Pneumonic consolidation	29 (52.72)	12 (41.38)	17 (58.62)
4.	Emphysema	19 (34.54)	07 (36.84)	12 (63.16)
5.	Chronic bronchitis	08 (14.54)	05 (62.50)	03 (37.50)
6.	Bronchopneumonia	07 (12.73)	04 (57.14)	03 (42.86)
7.	Tuberculosis	04 (25.45)	01 (25.00)	03 (75.0)
8.	Carbon Pigmentation	14 (25.45)	09 (64.29)	05 (35.71)
9.	Pulmonary alveolar fibrosis	04 (7.27)	03 (75.0)	01 (25.00)

Table XI shows geographical area wise distribution of pulmonary histopathological findings observed. Pulmonary congestion was seen mostly in rural cases (58.49%), remaining 41.51% cases belonged to urban area. Out of 48 cases (87.27%) of Pulmonary edema, it was observed in 29 cases (60.42%) of rural area and in 19 cases (39.58%) of urban area. Pneumonic consolidation was encountered in 17 cases (58.62%) of rural are and in 12 cases (41.38%) of urban

area. Out of total 19 cases (34.54%) of Emphysema observed, it was seen in 12 cases (63.16%) of rural area and 7 cases (36.84%) of urban area. Total 8 cases (14.54) of chronic bronchitis included 5 cases (60.25%) from urban area and 3 cases (39.75%) from rural area. Bronchopneumonia was found in 4 cases (57.14) of urban area and in 3 cases (42.86%) of rural area. Regarding Tuberculosis out of total 4(7.27%) cases, 3 cases (75%) were belonged to rural area and one case (25%) belonged to urban area. Increase carbon pigmentation was observed in 14 cases (25.45%) which included 9 cases (64.29%) from urban area and 5 cases (35.71%) from rural area. Pulmonary alveolar fibrosis was seen in 4 cases (7.27%) which included 3 cases (75%) of urban area and one case (25%) of rural area.

TABLE XII

Histopathological findings in relation to age and sex

S. No.	Histopathological Findings	Total No. of cases (%)	Age in Years											
			11 - 20		21 -30		31 - 40		41 - 50		51 - 60		61 - 70	
			M	F	M	F	M	F	M	F	M	F	M	F
1.	Pulmonary Congestion	53 (96.36)	4 (7.55)	4 (7.55)	4 (7.55)	9 (16.98)	10 (18.87)	6 (11.32)	9 (16.98)	2 (3.77)	3 (5.66)	-	-	2 (3.77)
2.	Pulmonary edema	48 (87.27)	4 (8.33)	2 (4.17)	4 (8.33)	8 (16.67)	9 (18.75)	6 (12.50)	9 (18.75)	1 (2.08)	3 (6.25)	-	-	2 (4.17)
3.	Pneumonic consolidation	29 (52.72)	4 (13.79)	3 (10.34)	3 (10.34)	6 (20.69)	2 (6.90)	3 (10.34)	4 (13.79)	1 (3.45)	2 (6.90)	-	1 (3.45)	-
4.	Emphysema	19 (34.54)	-	-	1 (5.26)	4 (21.05)	5 (26.32)	2 (10.53)	7 (36.84)	-	-	-	-	-
5.	Chronic bronchitis	08 (14.54)	-	-	-	-	2 (25.00)	-	3 (37.5)	1 (12.5)	2 (25.00)	-	-	-
6.	Bronchopneumonia	07 (12.73)	1 (14.29)	-	-	2 (28.55)	1 (14.29)	1 (14.29)	-	1 (14.29)	1 (14.29)	-	-	-
7.	Tuberculosis	04 (25.45)	-	-	-	3 (75)	-	-	1 (25)	-	-	-	-	-
8.	Microthrombi	14 (25.45)	3 (21.43)	1 (7.14)	1 (7.14)	3 (21.43)	3 (21.43)	1 (7.14)	1 (7.14)	1 (7.14)	-	-	-	-
9.	Bronchiolar epithelial change	14 (25.45)	2 (14.29)	2 (14.29)	-	4 (28.57)	4 (28.57)	1 (7.14)	-	1 (7.14)	-	-	-	-

Contd.

Table XII Contd.

S. No.	Histopathological Findings	Total No. of cases (%)	Age in Years: 11 - 20 M	11 - 20 F	21 -30 M	21 -30 F	31 - 40 M	31 - 40 F	41 - 50 M	41 - 50 F	51 - 60 M	51 - 60 F	61 - 70 M	61 - 70 F
10.	Carbon Pigmentation	14 (25.45)	1 (7.14)	1 (7.14)	-	3 (21.43)	2 (14.29)	1 (7.14)	4 (28.57)	1 (7.14)	1 (7.14)	-	-	-
11.	Thickened alveolar septa	07 (12.73)	1 (14.29)	-	1 (14.29)	1 (14.29)	2 (28.55)	1 (14.29)	-	1 (14.29)	-	-	-	-
12.	Calcification of vessels	05 (9.09)	-	-	-	-	-	-	1 (20)	1 (20)	1 (20)	-	-	2 (40)
13.	Pulmonary alveolar fibrosis	04 (7.27)	-	-	-	-	-	-	2 (50)	-	1 (25)	-	-	1 (25)

* Figures mentioned in ( ) shows percentage(%).

Table XII shows histopathological findings in relation to age & sex. Pulmonary congestion was mostly observed in 31-40 years age range, total 16 cases (30.19%) which included 10 males (18.87%) and 6females (11.32%), followed by 13 cases (24.53%) in 21-30 years age range including 4 males (7.55%) and 9 females (16.98%). 11 cases (20.75%) were observed in 41-50 years age range included 9 males (16.98%) and 2 females (3.77%).8 cases (15.1%) were found in 11-20 years age range with 4 males (7.55%) and 4 females (7.55%). 3 cases (5.66%) were observed in 51-60 years age range , all were males , and 2 cases (3.77%) were found in 61-70 years age range, all were females. Thus out of 53 cases (96.36%) of pulmonary congestion 30 cases (56.60%) were males and 23 cases (43.40%) were females.

Out of total 48 cases (87.27%) of pulmonary edema, most of the cases from 31-40 years age range 15 (31.25%) including 9 males (18.75%) and 6 females (12.50%), followed by 12 cases (25%) from 21-30 years age range including 4 males (8.33%) and 8 females (16.67%). 10 cases)20.83%) were observed in 41-50 years age range with 9 males (18.75%) and one female (2.08%). 6 cases (12.5%) were observed in 11-20 years age range including 4 males (8.33%) and 2 females (4.17%). 3 male cases (6.25%) were observed in 51-60 years age range and 2 female cases (4.17%) were found in 61-70 years age range. So out of 48 cases (87.27%) of pulmonary

edema , males were invalved in 29 cases (60.42%) and female were in 19 cases (39.58%).

Out of total 29 cases (52.72%) of Pneumonic consolidation 9 cases were observed in 21-30 years age range including 3 males (10.34%) and 6 females (20.69%), followed by 7 cases (24.13%) from 11-20 years age range including 4 males (13.79%) and 3 females (10.34%).5 cases (17.24%) were observed in 31-40 years and 41-50 years age range each, including 2 males (6.9%) and 3 females (10.34%) in 31-40 years age range and 4 males (13.79%) and one female (3.45%) in 41-50 years age range. Two cases (6.90%) were observed in 51-60 years age range , all were males and one female cases (3.45%) was found in 61-70 years age range. So out of total 29 cases (52.72%) of pneumonic consolidation, 16 males (55.17%) and 13 females (44.83%) were observed.

Emphysema was mostly observed in 31-40 years and 41-50 years age range including 7 cases (36.84%) from each. There were 5 males (26.32%) and 2 females (10.53%) in 31-40 years age range and it was present in 7 males (36.84%) only in 41-50 years age range. So out of 19 cases (34.54%) of emphysema it was observed in 13 males (68.42%) and in 6 females (31.58%).

Regarding chronic broncitis, maximum 4 cases (50%) were found in 41-50 years age range which included 3 males

(37.5%) and one female (12.5%), followed by 2 cases (25%) from 31-40 years and 51-60 years age range each included all male cases. So out of 8 cases (14.54%) of chronic bronchitis, it was observed in 7 males (87.5%) and in one female (12.5%).

Bronchopneumonia was observed in 2 cases (28.55%) in 21-30 years age and 31-40 years age range each , followed by one case each from 11-20 years , 41-50 years and 51-60 years age range respectively. All were females in 21-30 years and 41-50 years age range and all were males in 11-20 years and 51-60 years age range. One male and one female were found in 31-40 years age range. Thus out of total 7 cases (12.73%) of bronchopneumonia, it was present in 3 males (42.84%) and in 4 females (57.16%).

Tuberculosis was observed in 4 cases (7.27%) which included 3 cases (75%) of 21-30 years age range , all were females, and one male case (25%) of 41-50 years age range. So tuberculosis was predominantly present in females with a male ; female ratio of 1 : 3.

Microthrombi were found in 14 cases (25.45%). Maximum number of 4 cases (28.57%) were encountered in 11-20 years, 21-30 years , and 31-40 years age range each, followed by 2 cases (14.28%) from 41-50 years age range. 3 males were present in 11-20 years and 31-40 years age range each, while one female from 11-20 years, 31-40 years and

41-50 years age range each. Microthombi were observed in 3 females in 21-30 years age range. So microthrombi were encountered in 8 males (57.14%) and in 6 females (42.86%) with a male to female ratio of 1.3 : 1.

Regarding Bronchiolar epithelial change, majority of cases (35.71%) belonged to 31-40 years age range, followed by 4 cases (28.57%) in 11-20 yars and 21-30 years age range each, and one case from 41-50 years age range. All cases were females in 21-30 years and 41-50 years age range and 2 females in 11-20 years age range and one female in 31-40 years and 41-50 years age range each.

There were 4 males (28.57%) in 31-40 years age range and 2 males (14.29%) in 11-20 years age range. So out of 14 cases (25.45%) of bronchiolar epithelial change, it was found in 6 males (42.86%) and in 8 females (57.14%).

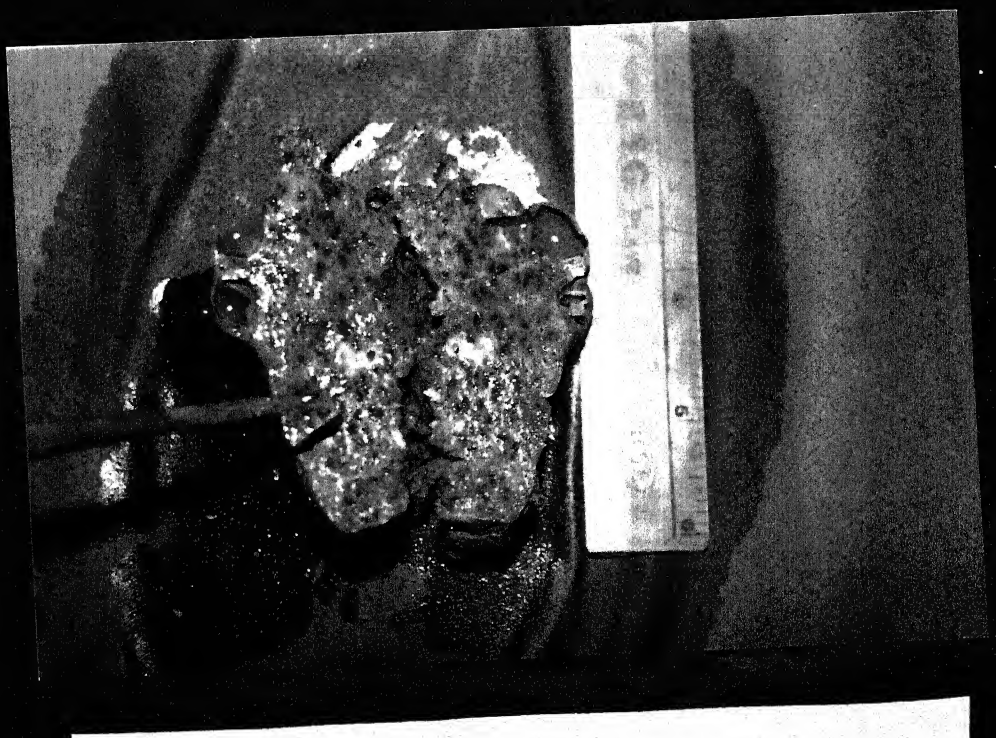
Increased carbon pigmentation was observed mostly in 41-50 years age range (35.71%), followed by 3 cases (21.43%) from 31-40 years and 21-30 years age range each, 2 cases (14.28%) from 11-20 years age range and one case from 51-60 years age range. Out of total 14 cases (25.45%) carbon pigmentation was observed in 8 males (57.14%) and in 6 females (42.86%).

Regarding, thickned alveolar septa most of the cases, were (42.84%) encountered in 31-40 years age range ,

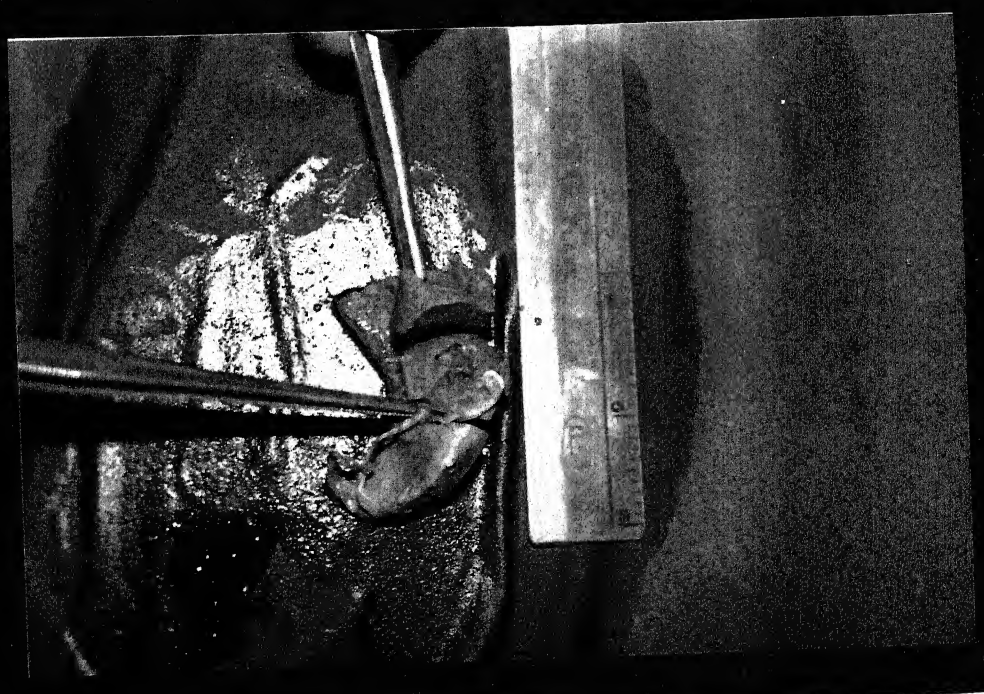
followed by 2 cases (28.58%) of 21-30 years age range and one case (14.29%) from 11-20 years and 41-50 years age range each. Out of total 7 cases (12.73%) , thickened alveolar septa were observed in 4 males (57.16%) and in 3 females (42.84%).

Calcification was observed in 5 cases (9.09%) , which included 2 cases (40%) from 61-70 years and 41-50 years range each and one case (20%) from 51-60 years age range ; with male to female ratio of 2 : 3.

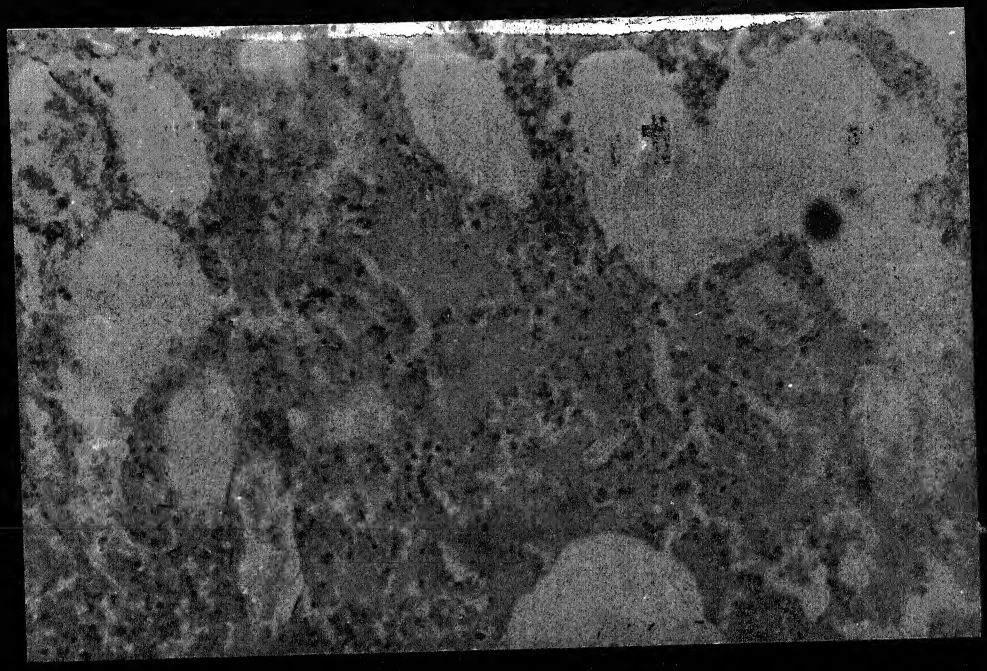
Pulmonary alveolar fibrosis was seen in 4 cases (7.27%) which included 2 cases (50%) from 41-50 yeas age rage and case from 51-60 years and 61-70 years age range each, with a male to female ratio of 3:1.



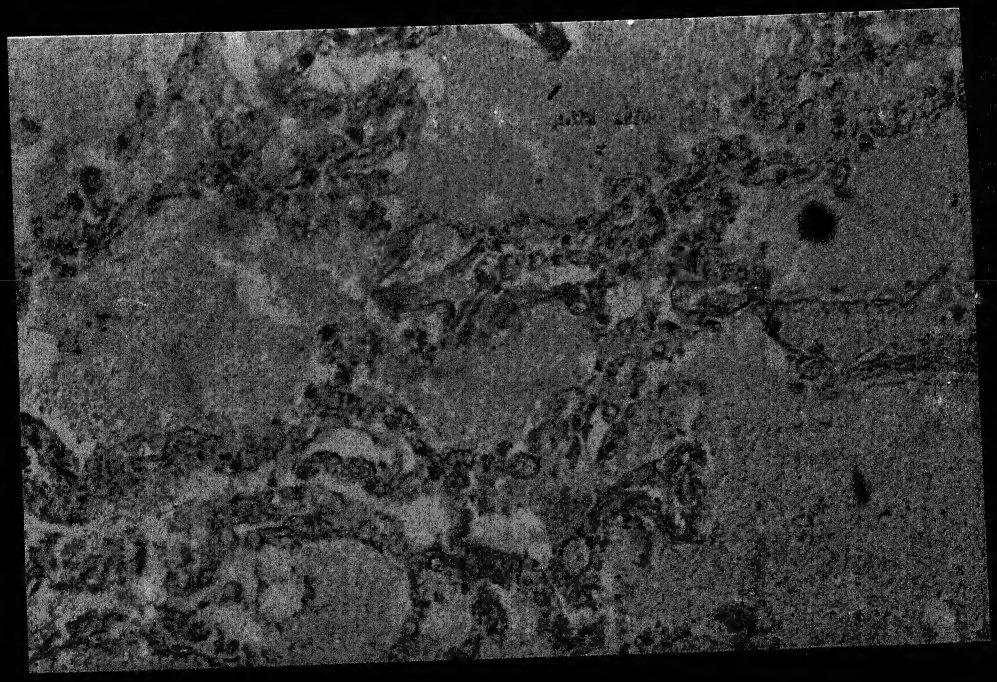
Photograph of lung specimen in gross showing cysts. (Forceps were used to display the lesion).



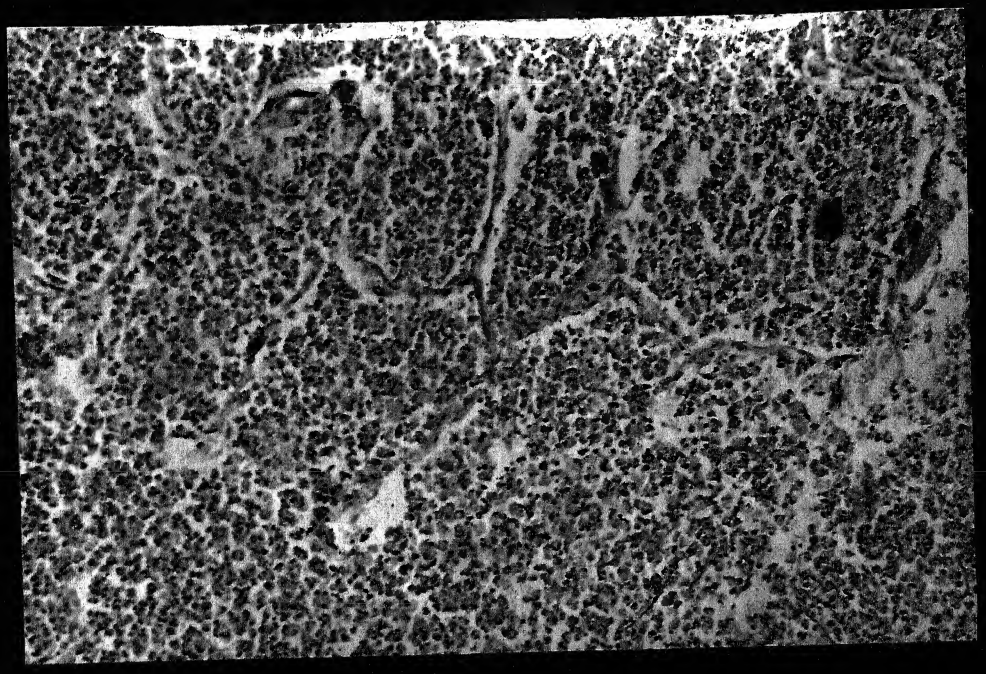
Photograph -Gross appearance of lung specimen showing calcification (Forceps were used to display the lesion).



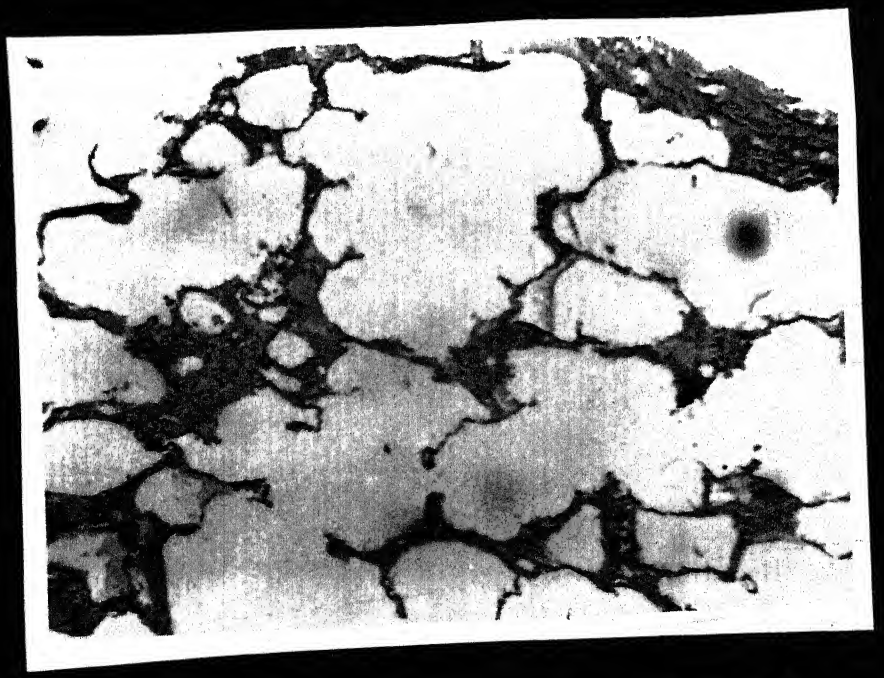
Photomicrograph showing Pulmonary congestion.
(H&Ex65)



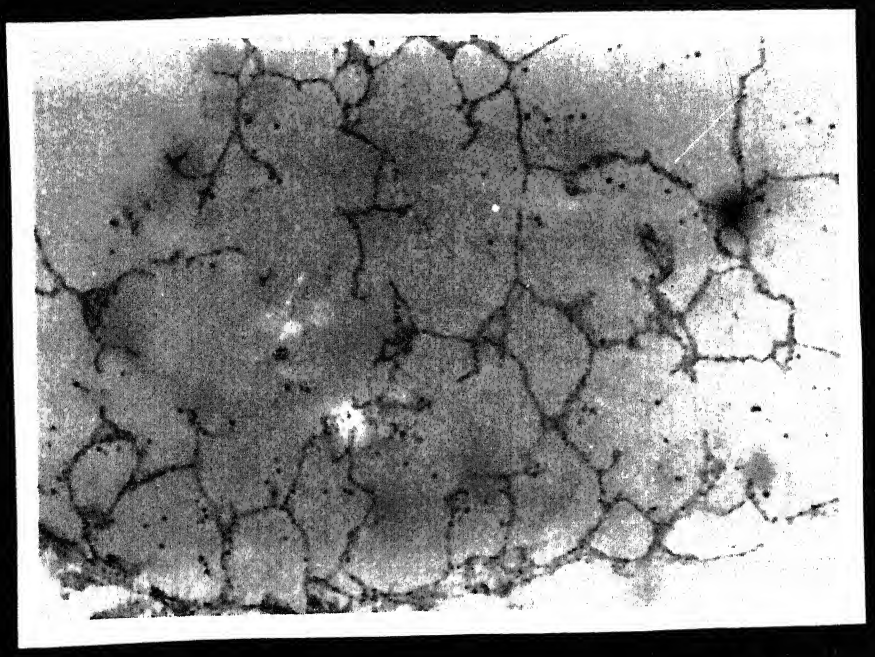
Photomicrograph showing Pulmonary edema.
(H&Ex65)



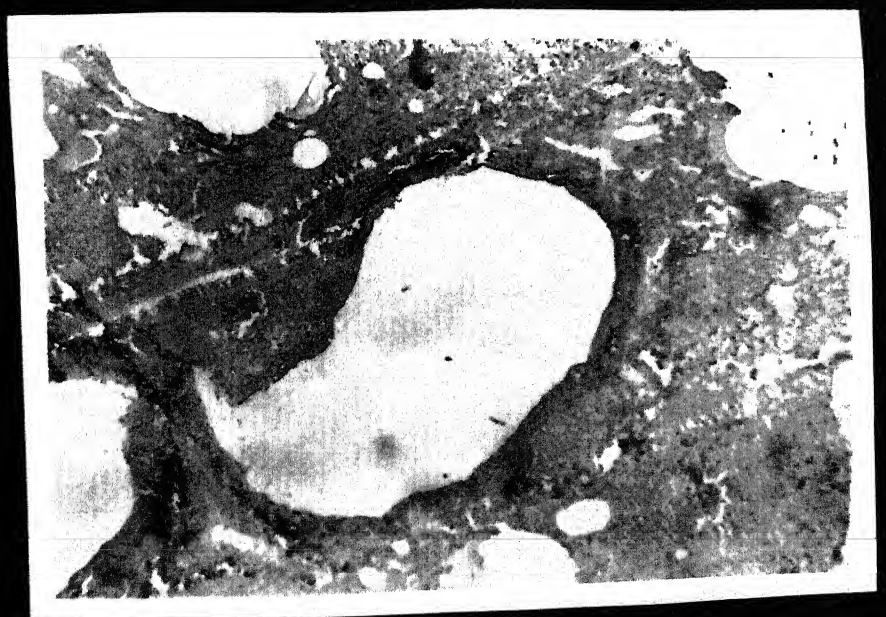
Photomicrograph showing Lobar pneumonia.
(H&Ex50)



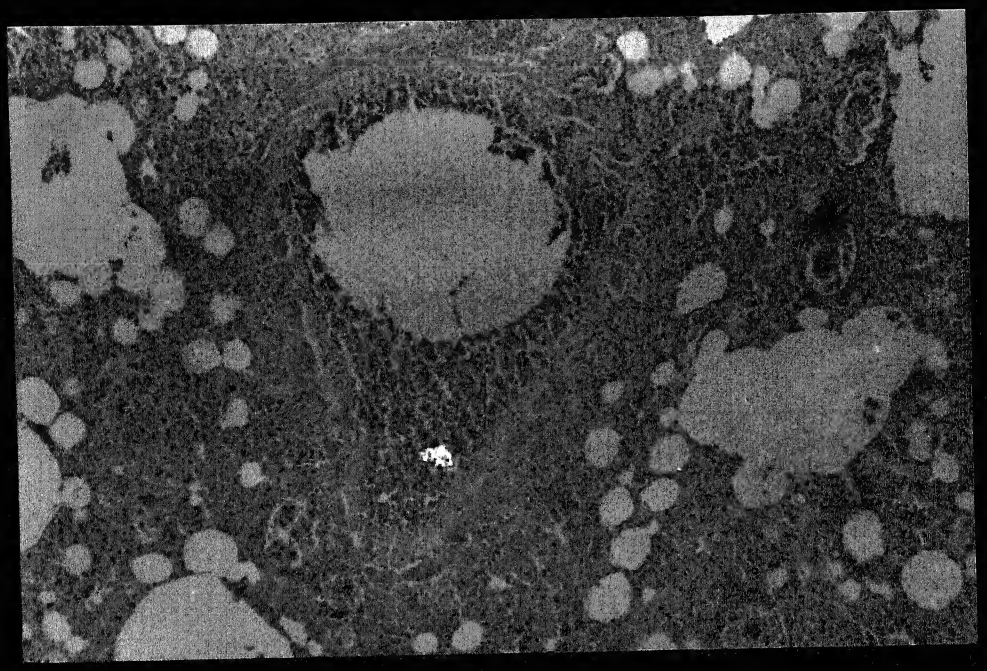
Photomicrograph showing Emphysema.
(H&Ex20)



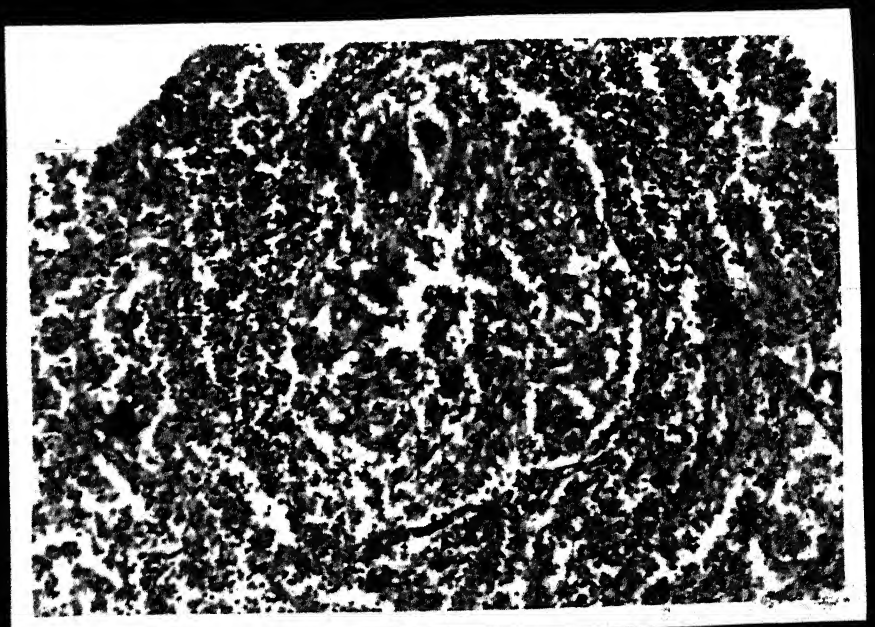
Photomicrograph showing Emphysema.
(H&Ex20)



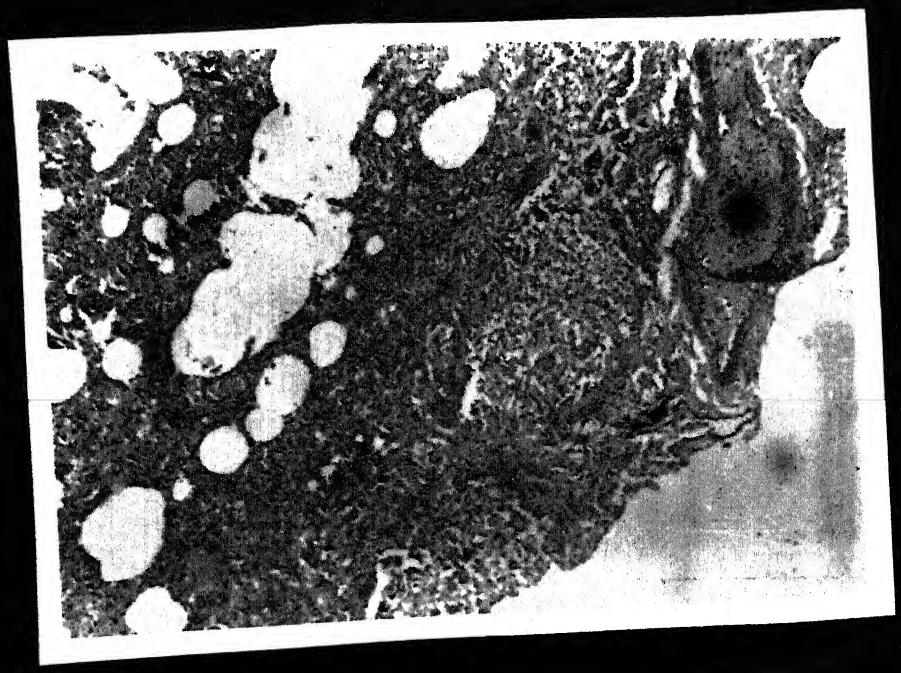
Photomicrograph showing Chronic bronchitis.
(H&Ex20)



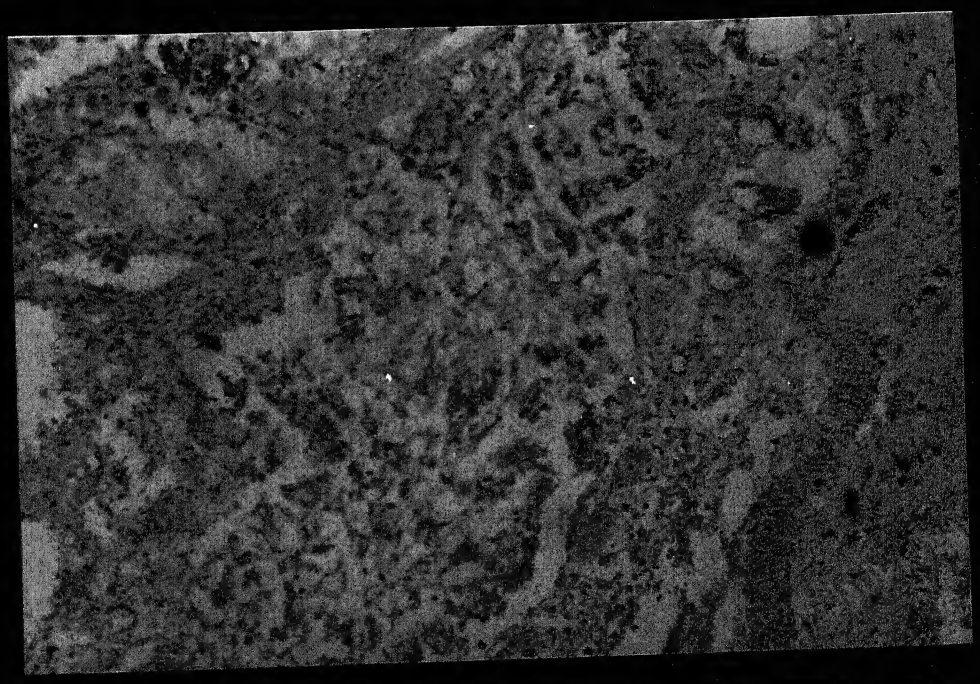
Photomicrograph showing Bronchopneumonia.
(H&Ex50)



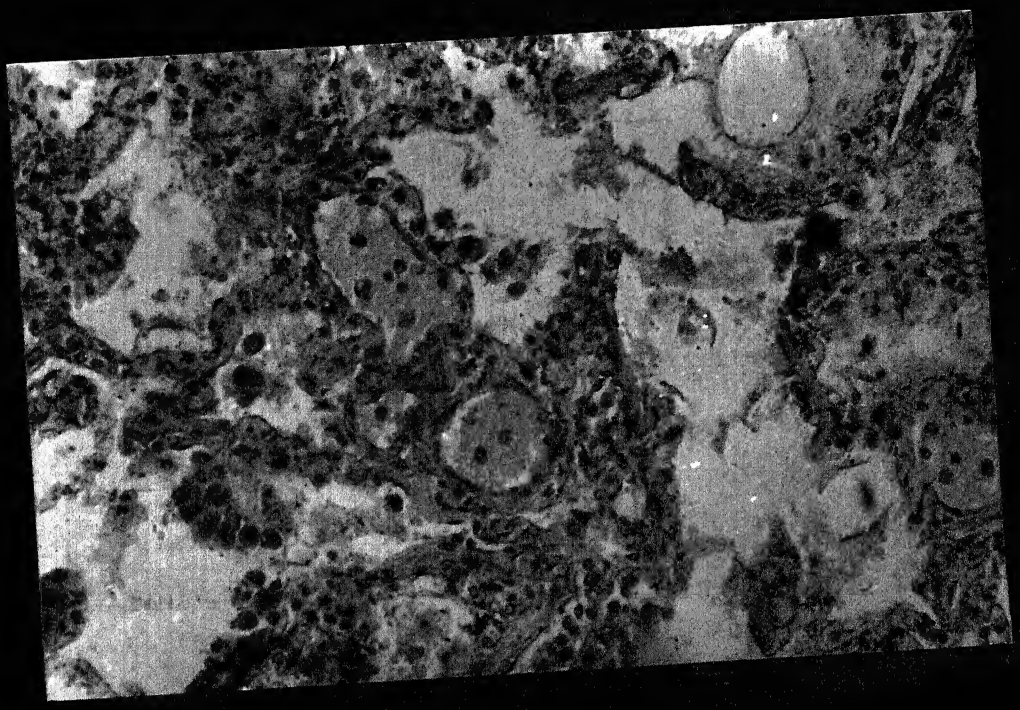
Photomicrograph showing Tuberculous granuloma.
(H&Ex50)



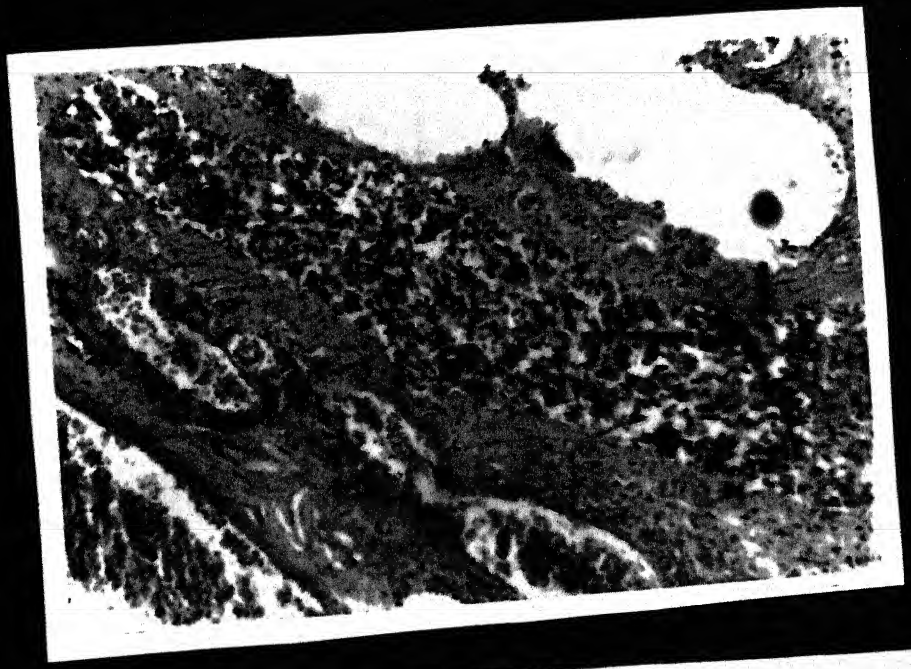
Photomicrograph showing Tuberculous granuloma.
(H&Ex20)



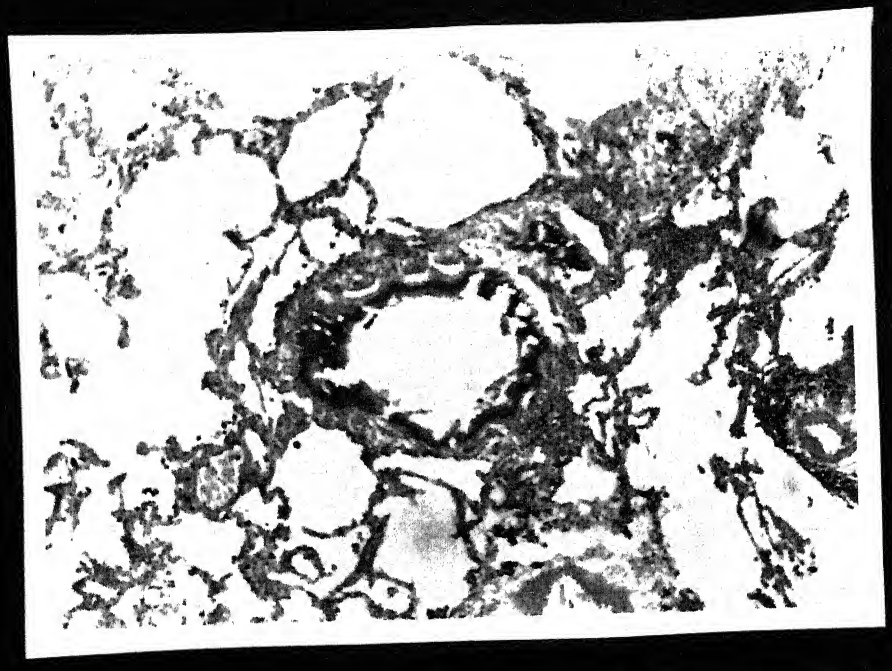
Photomicrograph showing Tuberculous granuloma.
(H&Ex65)



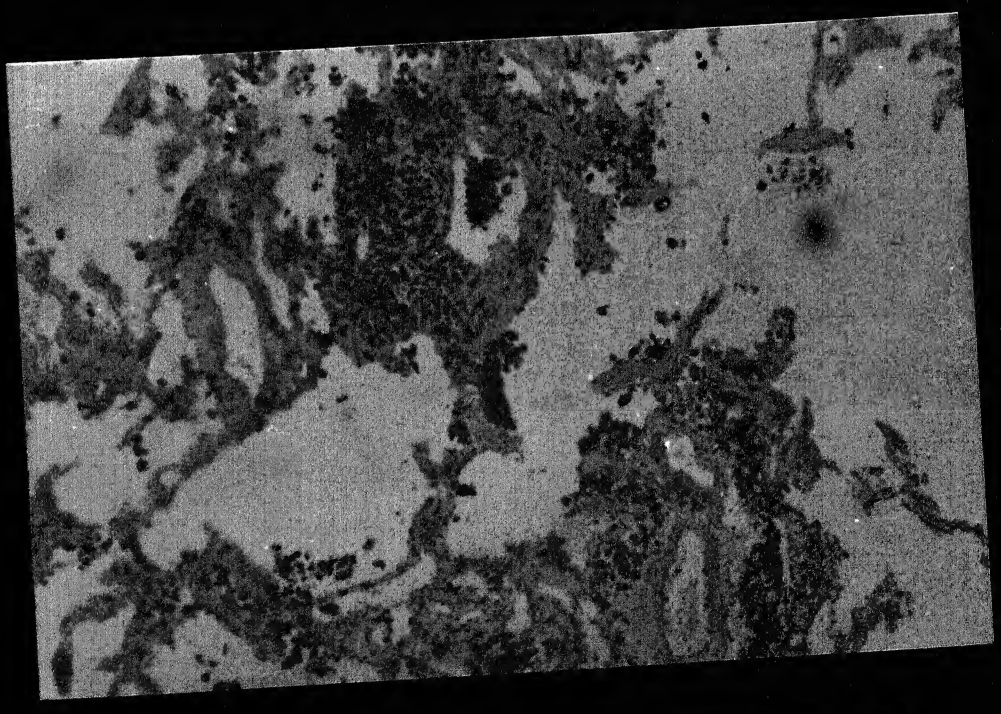
Photomicrograph showing Microthrombi. (H&Ex65)



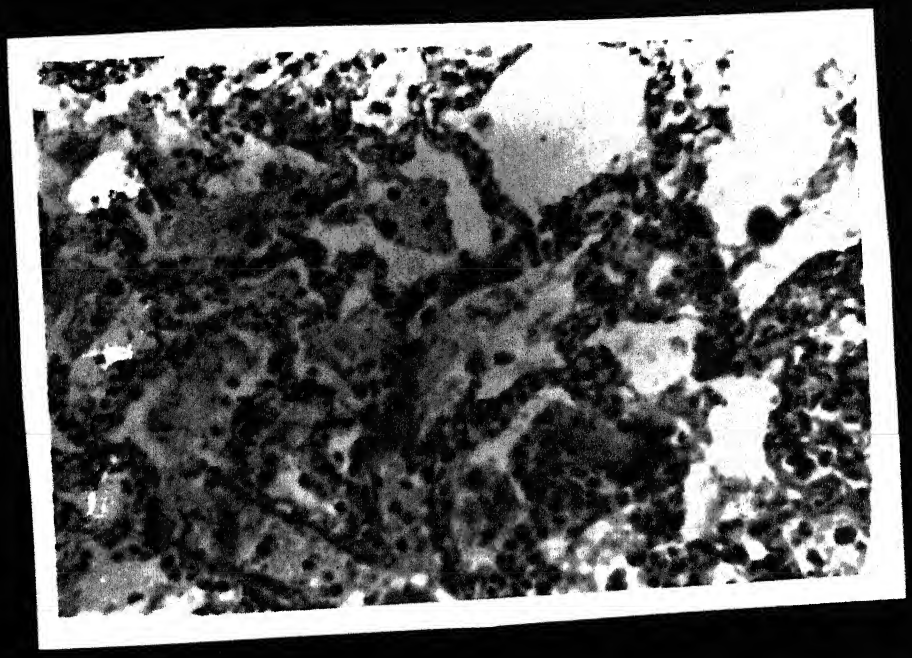
Photomicrograph showing Hyperplastic change of bronchiolar epithelium. (H&Ex50)



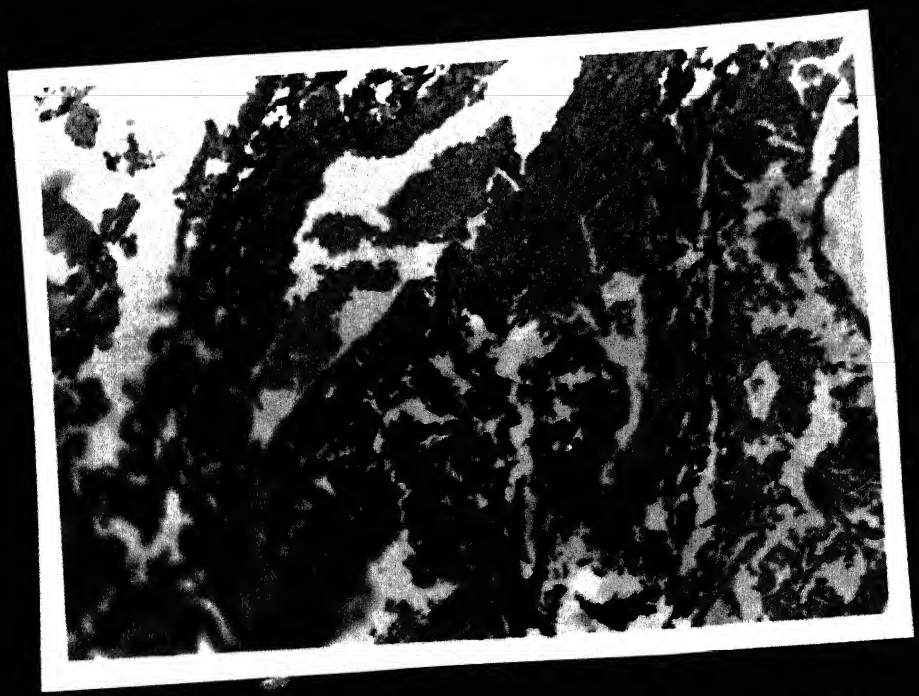
Photomicrograph showing Metaplastic change of bronchiolar epithelium. (H&Ex50)



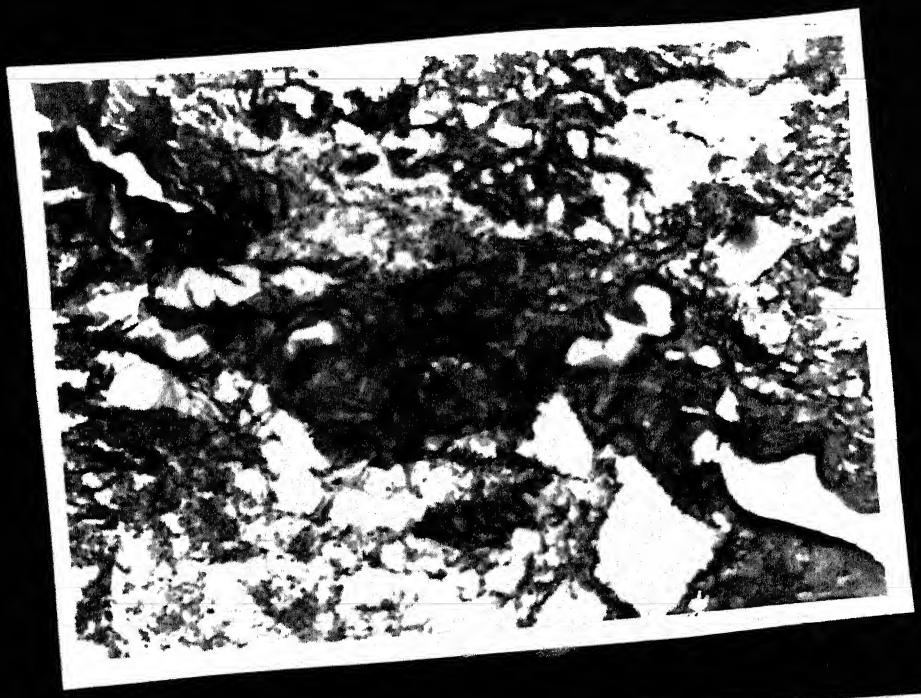
Photomicrograph showing carbon pigmentation. (H&Ex65)



Photomicrograph showing Thickened alveolar septa.
(H&Ex50)



Photomicrograph showing Calcification.
(H&Ex20)



Photomicrograph showing Pulmonary alveolar fibrosis.
(H&Ex20)

DISCUSSION

DISCUSSION

It is impossible to overemphasize the importance of lung diseases in the overall perspective of pathology and clinical medicine. Primary respiratory infections, such as bronchitis, bronchopneumonia and other forms of pneumonia, are common place in clinical and pathological practice. In this day of cigarette smoking and air pollution, chronic bronchitis and emphysema have become rampant, affecting large segments of the total population. Moreover, the lungs are secondarily involved in almost all forms of terminal diseases, so that at virtually every autopsy, some degree of pulmonary edema, atelectasis or bronchopneumonia is found.

A total of 55 autopsies were conducted in the mortuary of M.L.B. Medical college and Hospital , Jhansi. Lung specimens were also removed from a case referred from Military Base Hospital , Cantt. Jhansi. Victims were mostly males as compared to females with an incidence ratio of 1.2:1. Most of the cases belonged to third and fifth decade as well as cases from rural areas predominated over urban areas with an incidence ratio of 1.4:1. These findings of ours could not be compared due to paucity of such information in literature.

The time interval between death and autopsy in general was 20 hours. The cause of death in most of the cases

autopsied was accidental whereas in some cases, cause of death was in the form of burn injuries, poisoning and unexplained medical illness.

Lung specimens removed at autopsy, on gross examination were found grayish black in color as well as it was reddish brown in some cases. So as the cut surface, in most of the cases, it was grayish black which was might be due to carbon deposition in urban dwellers. Cyst and calcification were also observed. The above gross findings again could not be compared due to unavailability of such kind of data.

Commonest histopathological lesions observed were pulmonary congestion and pulmonary edema followed by pneumonic consolidation, emphysema, carbon pigmentation microthrombi, bronchiolar epithelial change, chronic bronchitis, bronchopneumonia, thickened alveolar septa, calcification of vessels, tuberculosis and pulmonary alveolar fibrosis.

As regard pulmonary congestion which was observed in variable severity ranging from mild to severe congestion in large number of cases. According to Parikh (1992), pulmonary congestion has been common finding on postmortem examination. Pulmonary congestion was more marked in bodies received from rural areas as compared to urban cases

which can be explained in certain forms on the basis of duration distance or time interval between postmortem and death. Considering congestion in relation to age, congestion has been common finding in persons after the age of 30 years which was predominant in male with a male to female ratio of 1.3:1. Such findings and observations could not be compared thoroughly due to paucity of such information in the literature.

Pulmonary edema of variable severity has been a consistent finding in large percentage of cases in our present study in the tune of 87.27%. Similar higher incidence has been reported by Agarwal et al (1983) in road traffic accident cases. Here again pulmonary edema was more commonly seen in persons after the age of 30 years, which was more preponderant in male with a male to female ratio of 1.5 : 1.

Pneumonic consolidation has been frequently encountered in approximately 52.72% cases with a high incidence in third decade and more prevalent in males as compared to females with a male to female ratio of 1.2:1. Present study shown bronchopneumonia in a good percentage of cases (12.73%), commonly afflicting persons in third and fourth decade of life and has been more common in females with a female to male ratio of 1.3:1. Bronchopneumonia finding was observed more frequently in urban as compared to rural areas. These findings were in accordance with Park

(1997) who has reported similar incidence of pneumonia (10-20%) in developing countries. Robbins (1989) stressed bronchopneumonia as a common postmortem finding in his series. According to Mac Gee W (1993), bronchopneumonia is very fetal in geriatric population.

Emphysema as a histopathological finding was encountered in 34.54% cases with male to female ratio of 2:1, predominating in rural as compared to urban population. Considering emphysema in relation to age, it was commonly observed in fourth and fifth decade of life. This can be easily explained on the basis of condition prevailing in rural population and also in urban population in high percentage. Our findings are in close accordance with the findings of Robbins (1989), and Anderson (1996) who has stressed that prevalence and severity of emphysema increases with age and greater in men than women. Anderson (1996) reported 20 - 100% prevalence and Thurlbeck (1976) reported 50% incidence of emphysema at autopsy.

In present study chronic bronchitis has encountered in 14.54% cases with male preponderance as a male to female ratio of 7:1, predominant in urban as compared to rural area. Chronic bronchitis was mostly observed in persons in fifth decade. The present findings are very similar to that of Robbins (1989), Davidson (1995) and Anderson (1996). Thurlbeck (1976) reported 10-25% of the urban adult

population have chronic bronchitis, may be due to community air pollution and industrial causes.

Bronchiolar epithelial change was observed in 25.45% cases which was mainly present in the persons below 40 years of age. In younger age group, bronchiolar epithelial change was observed due to burn injuries and in later life, it may be due to smoking habits, air pollution and industrial causes. These findings and observations could not be compared because of unavailability of such findings in literature.

Increase carbon pigmentation has been also encountered in good percentage of cases (25.45%), with male predominance having male to female ratio of 1.3:1, mostly seen in urban population than rural area. Maximum number of cases were seen in fifth decade of life. These findings and observations are in accordance with Robbins (1989), who stressed that carbon pigmentation is more common in urban dwellers due to air pollution and industrialization.

As regarding tuberculosis, it was observed in 7.27% cases with female predominance having female to male ratio of 3:1, and predominantly encountered in rural areas as compared to urban population. Most of the cases were observed in third decade followed by fifth decade of life. These findings are in accordance with Park (1997) who reported that

tuberculosis is more common among rural areas, in elderly males and in females below 35 years age.. In present study tuberculosis was found in 7.27% cases while according to W.H.O. survey (1997), for India prevalence rate of tuberculosis is 30% and incidence rate is 1-2%. The difference is because our study is autopsy study and we have studied less number of cases.

Microthrombi were observed mostly in persons dying of burn injuries, predominated in males with an incidence of 25.45%. According to Sevitt S. (1966), presence of microthrombi as well as macrothrombi are common findings in lung in persons dying of burn injuries which can be also a cause of death in such cases.

Thickened alveolar septa was observed in 12.73% cases with male predominance having male to female ratio of 1.3:1, mostly in fourth decade of life. These findings again could not be compared due to lack of such information in the literature.

In our study calcification was observed in 9.09% cases with female predominance with a female to male ratio of 1.5:1, mostly in seventh and fifth decade of life. These findings are in accordance with Anderson (1996), that lung is one of the favoured sites of calcification.

Regarding pulmonary alveolar fibrosis, which was found in 7.27% cases with male predominance as a male to female

ratio of 3 :1. It was mostly observed in persons after the age of 50 years. These findings are very similar to Robbins (1989) and Anderson (1996) who stressed that pulmonary fibrosis can occur at any age but mostly occurs in middle aged or older persons.

The present study has been a small one comprising few cases in a short period of time and hence the limited findings were observed. It is suggested that in future, such kind of study should be undertaken in larger perspective including large number of cases so that the findings and observations can be of much utility and use and the study should have become more fruitful.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

The present study entitled "Study of Lung Diseases in Bundelkhand Region of U.P. (A Potmortem study)" conducted in the Department of pathology, M.L.B. Medical College and Hospital, Jhansi, Comprises analysis of total of 55 autopsies from mortuary of M.L.B. Medical College & Hospital, Jhansi, also included one autopsy case from the Military Base Hospital, Cantt. Jhansi.

Victims were aged between 11 years to 65 years with predominance of males as compared to females with ratio of 1.2:1. Majority of cases belonged to rural areas of Bundelkhand region of U.P.

Different histopathological lesions were encountered included Pulmonary congestion (96.36%), Pulmonary edema (87.27%), Pneumonic consolidation (52.27%), Emphysema (34.54%), Carbon pigmentation (25.45%), Microthrombi (25.45%), Bronchopneumonia (12.73%), Thickened alveolar septa (12.73%), Calcification of vessels (9.09%), Tuberculosis (7.27%) and Pulmonary alveolar fibrosis (7.27%).

It is concluded that the present study is a small one comprising few cases in a short period of time and hence the limited findings were observed. It is suggested that in future such kind of study should be undertaken in larger perspective including large number of cases so that the findings and observations can be of much utility and use and the study should have become more fruitful.

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